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DESTRUCTION OF THE CEREBRAL CORTEX FOLLOWING NITROUS OXIDE-OXYGEN ANESTHESIA*

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NITROUS oxide-oxygen anesthesia is relatively safe, although fatalities have been reported by several authors. So far as we could ascertain Baldwin[1] was the first to call attention to the harmful effects of nitrous oxide. He described 14 fatalities; Caine[2] reported four; Davies[3] one; Hahn[4] two, and McKean Downs[5] six cases. Caine[2] was the first to consider brain damage as the possible cause of death, but no histologic evidence was given by any of the above authors. We are reporting three cases in which there was destruction of the cortex and basal ganglia following the use of nitrous oxide-oxygen anesthesia and one case with clinical evidence of a similar process.

CASE REPORTS

Case 1.—R. B., white male, aged 22, was cut on the finger by a piece of metal. Treatment was begun October 24, 1932. Examination showed a small wound which would admit a grooved director. There was a slight purulent discharge and swelling of the finger. Temperature was 99.2° F., pulse 85. On October 25, under local anesthesia, the wound was enlarged and drained and the swelling subsided. On November 1 the swelling recurred and the wound was again enlarged. About 4 cc. of pus were removed and better drainage established. Nitrous oxide-oxygen anesthesia was employed for the second operation.

Twenty minutes after the beginning of the administration of anesthesia, respiration suddenly ceased and there was rigidity and tremor of the extremities. Oxygen was administered and respiration was reestablished ten minutes later, but it was forced and 40 per minute. The patient remained unconscious from that time until death. The temperature rose rapidly to 107° F. and ran a very irregular course, ranging from 103° to 107°. The pulse varied from 120 to 160, roughly following the temperature curve. The blood pressure varied between 85/60 and 125/60. All tendon reflexes disappeared permanently. There were periods of increased rigidity and tremor of the entire body when the temperature rose, but between these periods there was a lead pipe

* The material from Cases 1, 2 and 3 was sent to us for study.

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rigidity of the extremities. The muscles of mastication were hypertonic with occasional relaxation. Pupils were equal and symmetrical, varying from normal width to wide dilation, and reacted well to light. During the last eight hours the eyes were turned to the right. The margin of the right disk was obscured and the veins were engorged; the left fundus was not seen. There was incontinence of urine and feces. Laboratory findings: The spinal fluid pressure was increased but was otherwise negative. The white count was 18,000. Urine was negative. Death occurred approximately 60 hours following operation.

Postmortem Examination.—Brain: Leptomeninges were thin and smooth and were distinctly hyperemic. The convolutions were well developed. The basal vessels were delicate. On frontal sections the gray matter was of normal width, well demarcated and contained numerous petechial hemorrhages, especially in the occipital lobes. The basal ganglia and brain stem were hyperemic and there were few scattered small hemorrhages. Ventricles were narrow and the ependyma smooth. The cerebellum appeared normal. The remainder of the postmortem was negative.

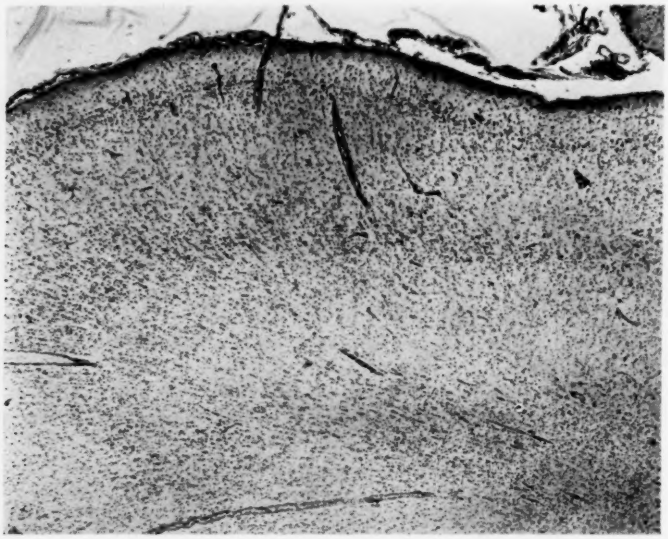

FIG. 1.—Section of parietal cortex Nissl stain. Photomicrograph Zeiss planar 20 Mm. The third, fifth and sixth layers are destroyed. The second and fourth layers are present, but show definite degenerative changes.

Histology.—Leptomeninges were thin and showed no pathologic changes other than hyperemia.

Cortex: (Nissl stain). There was a very severe disturbance of the laminar type. The first layer contained a few scattered astrocytes; the second layer was very thin, the neurons being greatly reduced in number and those remaining were degenerated. The third layer was entirely destroyed, and contained only a few degenerated neurons. The fourth layer was better preserved and contained a considerable number of ganglion cells. The fifth and sixth layers were completely destroyed and many areas were transformed into a spongy state (Fig. 1). The cortical myeloarchitecture also showed severe changes. The tangential fibers were almost entirely destroyed, but the radial fibers were somewhat better preserved. Silver preparation showed a reduction of axis cylinders.

Basal Ganglia.—Caudate and Putamen: The parenchyma was severely affected, the small neurons being almost completely destroyed, but the larger ones were better preserved. There were large areas of spongy state in the putamen.

Pallidum, Thalamus and Hypothalamus: The parenchyma showed severe degenerative changes, but the cells were only moderately reduced in number.

Midbrain: In the corpora Luys, geniculate bodies, red nuclei, central gray substance and reticulate substance there was moderate degeneration of the neurons but no reduction in their number. The substantia nigra and the pons were normal.

Medulla: The neurons of the large olives were shrunken but otherwise there were no changes.

Cerebellum: The Purkinje elements were destroyed in many areas, but the granular and molecular layers were intact.

The cytologic changes of the neurons were of two types (a) shrinkage, (b) ischemic necrosis:

(*a*) The shrunken cells appeared small, with pyknotic nuclei and greatly reduced cytoplasm. They were devoid of tigroid substance and were frequently reduced to shadows.

(*b*) The ischemic elements were irregularly shaped or elongated, the cytoplasm was pale, homogeneous or slightly granular, the nuclei being stained a deep blue.

The first type was particularly frequent in the cortex, putamen and caudatum, the second in the pallidum and thalamus.

The glial response was moderate, being restricted to scattered microglia and macroglia. The blood vessels showed a slight capillary proliferation. Perivascular hemorrhages were present in some of the areas. Lipoids and iron were not present.

White matter: the oligodendroglia was increased and swollen. The blood vessels were slightly proliferated and there were scattered perivascular hemorrhages. There was no evidence of demyelinization.

Case 2.—W. G., a white male, aged 28, was admitted July 1, 1934, complaining of intermittent attacks of pain in the right lower quadrant, associated with nausea and vomiting. There were no other complaints.

Operation July 2, 1934. A hyoscin-morphine-cactoid tablet No. 1 was administered hypodermically one hour before operation. Nitrous oxide-oxygen anesthesia was administered with the McKesson Nargraf apparatus. The patient had an easy induction and the anesthesia proceeded smoothly for approximately 30 minutes, when it was noted that respiration became quieter and more shallow. Oxygen was administered freely and finally under pressure. In spite of this, respiration continued to become more quiet and finally ceased. For a period of 40 minutes the patient apparently did not breathe voluntarily. Artificial respiration was continued and carbon dioxide added to the oxygen. Stimulants such as Alpha lobelin, caffeine, atropin and coramine were administered without effect. The blood pressure and the pulse rate which were normal at the beginning of the anesthesia rose to 150/90 and 120 respectively, the latter being full and of good volume. The color of the skin was pink and the patient was apparently in good condition except that he did not breathe. The pupils were moderately dilated and did not react to light, the muscles were relaxed.

After 40 minutes the patient began breathing, at first taking a few shallow breaths, which gradually became deeper. Oxygen-carbon dioxide was administered for two hours. The patient remained comatose and developed stiff neck and convulsive movements of the jaw, biting tongue and lips, and was very restless. In the afternoon his temperature rose to 105.6°, pulse to 145 and respirations to 24. Two days later he showed signs of returning consciousness and asked for water. Twitchings of the left index and third fingers were noted. About 60 hours after the operation the respirations rose to 60; the pulse became very irregular in rhythm and volume; the temperature rose to 106° and there was deep coma. Death occurred approximately 72 hours after operation. Postmortem disclosed no gross findings of importance.

Histology.—Leptomeninges were normal. The entire cortex and the basal ganglia were severely damaged. The findings varied considerably in different areas of the cortex as follows:

(1) Areas in which all layers of the cortex with the exception of the second were destroyed, the parenchyma being reduced to a few scattered degenerated neurons.

(2) Areas in which the second and third layers were preserved while the fourth, fifth and sixth were destroyed.

(3) Areas in which the destruction was confined to the fifth and sixth layers (Fig. 2).

(4) Areas in which the destruction of the parenchyma was spotty.

Spongy state was present throughout the cortex, occurring usually in the fifth and sixth layers. The axis cylinders were considerably reduced in number. Myeloarchitectural changes corresponded to those of the first case.

Basal Ganglia: the parenchyma of caudatum, putamen, pallidum and thalamus was destroyed.

Midbrain, Hypothalamus, Medulla and Cerebellum: the changes corresponded to those in the first case. The reticulate substance showed a moderate reduction of neurons, with severe degenerative changes of the cellular structure.

Cytologic Changes: The degenerative changes of the neurons were of two types: (1) simple shrinkage, (2) ischemic degeneration with encrustations. The shrinkage

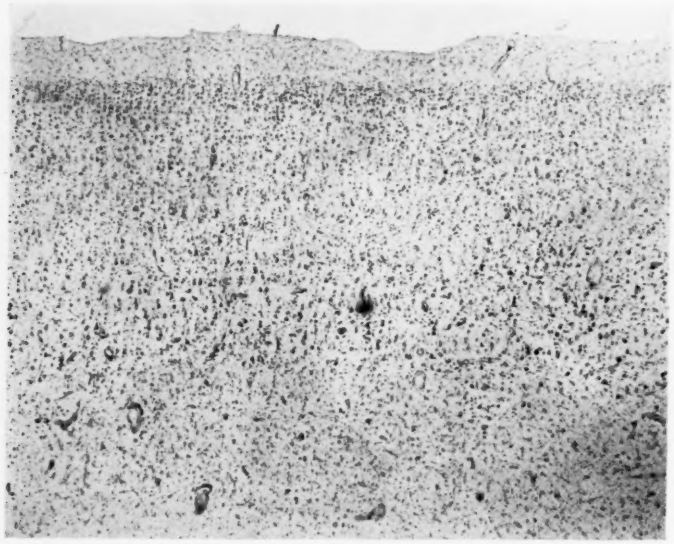

FIG. 2.—Section of parietal cortex Nissl stain. Photomicrograph Zeiss 20 Mm. The fifth and sixth layers are destroyed, the other layers show moderate diffuse degenerative changes.

corresponded to that of Case 1. The encrustations appeared in the form of numerous deep blue or black granules in the cytoplasm and on the dendrites of the cell. This change was common in the cortex but rare in the basal ganglia.

Blood vessel proliferation was marked in the basal ganglia, particularly in putamen and caudatum. There were no hyperemia or perivascular hemorrhages. Glial reaction corresponded to that of Case 1. Lipoid and iron stains were negative.

White Matter: the findings were identical with those in Case 1.

Case 3.—C. K., a white female, aged 51, admitted July 12, 1932, with complaints of menorrhagia and leukorrhea. Her health was otherwise good. General physical examination negative. Pelvic examination revealed a large, nodular mass. Diagnosis, uterine fibroid.

Operation July 13, 1932. Anesthesia was induced quietly. Panhysterectomy was performed. One hour later, while closing the wound, respiration and pulse suddenly ceased. Oxygen was given under pressure, adrenalin was injected intravenously, which effected prompt restoration of the pulse and respiration. (Time and details not mentioned.)

Following the operation the patient remained comatose and her entire body was rigid, and there was "risus sardonicus." Temperature, 100.9°; pulse, 100; respiration, 20; blood pressure, 140/80. The next morning (July 14) the temperature rose to 102.5°, the pulse to 120, but it was regular and of good volume. The patient perspired profusely. In the afternoon the respiration became very slow—eight per minute—and remained so for approximately 12 hours. The muscles of the extremities relaxed but the trunk muscles remained rigid.

During the next three days the temperature ranged between 104° to 105°, the respiration from 28 to 36, the pulse 110 to 120, blood pressure averaged 149/68. The patient continued comatose and incontinent. The entire body was rigid and there were twitchings of the left hand. On July 18 the pulse rose to 160 and became irregular; the respiration became very shallow, ranging between 32 and 40, ceasing for one to one and one-half minutes at intervals. The patient became increasingly cyanotic. The temperature rose to 106.4°. Several hours before death respiration gradually slowed down from 42 to 16 per minute. Death occurred July 19, approximately 119 hours following operation.

Postmortem revealed no gross findings of importance.

The histologic findings corresponded to those in the first two cases. The destruction involved the entire cortex and was of several types as follows:

(1) Areas in which mainly the upper layers (two and three or three and four) were damaged.

(2) Areas in which mainly the lower layers (four and six or five and six) were affected.

(3) Areas in which the entire width of the cortex was involved.

(4) Areas in which the destruction was spotty.

Spongy state was present in numerous areas.

In the basal ganglia, hypothalamus, brain stem, pons, and cerebellum no definite focal lesions were found, but the neurons showed moderate nonspecific changes.

The histologic details in the cortex and white matter corresponded to those of Cases 1 and 2.

Histologic Summary: In spite of some variation in degree the histologic picture of the three cases was essentially the same. There was severe damage throughout the cortex especially in the fifth and sixth layers (Case 2). In the first and third cases there were many areas in which the entire cortex was destroyed. The spongy state was usually restricted to the lower layers even in areas of diffuse destruction.

The damage of the basal ganglia was marked in Cases 1 and 2 and moderate in Case 3. The changes in the brain stem and cerebellum were in all cases much less severe than those of the cortex and basal ganglia. The histologic picture was purely degenerative in type.

Case 4.—J. D., white female, aged 29, admitted September 9, 1928, with a lung abscess. Phrenectomy was performed under local anesthesia with considerable improvement. The patient was discharged October 19, 1928. The symptoms recurred and the patient was readmitted November 16, 1928. The abscess was opened with cautery under local anesthesia and the cavity drained and packed with vaseline gauze. There was a rapid improvement and the patient was discharged. Eight months later the patient returned for closure of the fistula. On October 3, 1929, a bronchial and musculocutaneous plastic operation was performed and several large hilar bronchi were closed. The operation was conducted under nitrous oxide anesthesia, and lasted one and one-half hours.

The operation and anesthesia were without incident. During the operation the patient received 900 cc. of 5 per cent glucose per rectum and a hypodermic of morphine sulphate gr. 1/6. The patient's condition before, during and immediately after the operation was reported as fair.

In the afternoon she became restless and was given Pantopon gr. 1/3. At 8 P.M. hyoscin gr. 1/100 was given. One hour later the patient developed "spasmodic jerkings"

of the legs which soon involved both upper and lower extremities. The slightest stimulus precipitated a convulsive attack. There was fecal and urinary incontinence. The next morning, the patient was much improved and quieter but there were still slight muscular twitchings in the arms and face. The temperature was 102.6°, pulse 134, respirations 24. The head was turned to the right, but there was no rigidity of the neck. The fundi showed slight papilledema. Tendon reflexes were normal. The abdominal reflexes were absent. There was a positive Babinski on the left and a suggestion of one on the right, and the muscle tone in the legs was slightly increased. Tendo achillis pressure caused an expression of pain but no movement of the large toe. In the afternoon the patient again became noisy and restless and was given 1/100 of a grain of hyoscin. Toward the evening she was semistuporous and irrational, but answered questions at times.

During the next eight days the general condition showed a slow improvement, but on October 12 the patient was still somewhat confused and complained of impaired vision. There was a definite limitation of outward rotation of both eyes and a fine nystagmus on lateral deviation to the right. In moving the eyes upward the left eyeball lagged behind the right and went upward in jerks. Pupils were moderately dilated and did not react to light. Nasal margins of both disks were blurred, but there was no measurable elevation. Apparently there was no light perception in the right eye, but with the left she could count fingers. There was weakness of the left side of the face. The biceps and triceps reflexes were normal on the right and overactive on the left. The patient did not move the left arm and hand. When asked to squeeze with the left hand she closed the right hand. The abdominal reflexes were absent, the knee jerks were normal on the right and increased on the left. The Achilles reflexes were normal on both sides. Babinski was positive on the left, questionable on the right. There was marked sweating of both feet.

During the next few days considerable improvement was noted. The patient became oriented and emotionally stable. A slight VIth nerve palsy persisted. There was coarse tremor of the tongue on protrusion; the face was mask-like. The left arm and hand were paralyzed and there was considerable ataxia of the right arm. Knee and Achilles reflexes were normal and there was no Babinski sign on either side.

On October 22, she could count fingers at arm's length and began to move the left hand, but the ataxia on the right persisted. On October 26, the fundi were normal. Both arms were ataxic and the tone of both legs was markedly increased, more so on the right. Tendon reflexes were increased and there was an abortive ankle clonus. The umbilical reflexes were absent. On October 31, the patient complained of severe sharp pain in the hands, especially the right, associated with numbness and tingling. Strength of both hands was good, better on the right. Movements on the right were carried out better than on the left. On November 5, the patient was able to read words of ordinary print. There was some improvement of the left leg, enabling her to take a few steps. On the next day severe pain occurred in both arms and legs, more so of the left arm and right leg. Movements of the extremities accentuated the pain.

On November 9, the patient was up in a wheel chair. The extra-ocular movements and fundi were normal. The tongue protruded straight, and there was only slight weakness of the face on the left. Grip in the right hand was good but there was a marked ataxia on the right and an intention tremor on finger to nose test on both sides. There was bilateral adiadokokinesis. Grip on the left was weak. The biceps and triceps reflexes were normal. There was spasticity and increased tone of the legs and the knee and Achilles reflexes were increased bilaterally. There was bilateral ankle clonus. Vibratory sense was lost in both ankles. The sense of motion and position was lost in the toes of both feet.

By November 27, the patient was able to write legibly. Visual fields showed concentric contraction, relative central scotomata in both eyes, and later on a homonymous defect in the right inferior quadrant. The ophthalmologic symptoms improved gradually

and on January 12, 1930, the scotomata were much smaller but the fields still showed concentric contraction.

At the time of discharge from the hospital May 3, 1930, the pupils were equal, but reacted sluggishly to light. There was a slight coarse tremor of the tongue, marked ataxia on finger to nose test, which was not markedly increased by closing the eyes, bilateral adiadokokinesis, and hyperactive tendon reflexes. There was slight spasticity with atrophy of the right leg, and heel to knee to toe test was impossible on both sides. On September 8, 1930, the patient was ataxic, but was able to walk. The neurologic status was otherwise unchanged.

The patient was seen last on March 12, 1935. The pupils, extra-ocular movements, fundi and visual fields were normal. There was slight weakness of the left face with some masking. The tongue protruded straight. Biceps and triceps reflexes were exaggerated, more so on the right. Hoffmann's sign was positive on the left, the abdominal reflexes were diminished and the knee and Achilles reflexes were exaggerated. There was a suggestive bilateral Babinski and ankle clonus. There was hyperalgesia of the organic type below the sixth dorsal segment, but no other sensory changes. There was marked ataxia on finger to nose test, more marked on the left, which was increased with the eyes closed. There was a definite Romberg's sign and a tendency to fall toward the right. There was a slight spastic-ataxic gait, more marked on the left. Mentality was not materially impaired.

Clinical Discussion.—At the present time it appears that the harmful results of nitrous oxide-oxygen anesthesia can be divided into two groups as follows: (1) Fatal cases, and (2) cases with incomplete recovery. The first group can be subdivided into (A) immediate death, and (B) those with death occurring after hours, days or weeks.

(A) Death during nitrous oxide-oxygen anesthesia has occurred following narcoses of varying duration. The shortest narcosis with a fatal result was reported by Davies[3]—20 seconds, in Baldwin's[1] Case 4 "just in the beginning of the narcosis," in his Case 9, after 20 minutes. McKean Downs[5] reported a fatality after 45 minutes; Hahn[4] reported two cases with fatal termination following an anesthesia of an hour and a quarter. In many instances the duration of the narcosis is not given, but it can be assumed that in some instances it was not long, since the anesthetic was used for minor operations as, for example, extraction of teeth.

According to all authors, in fatal cases the respiration ceases suddenly and without warning (Baldwin, Caine[2], Davies, McKean Downs, Hahn[4]). McKean Down states: "There was no gasping, no change in the rate or depth of respiration, no futile efforts to breathe—merely a sudden and complete respiratory failure . . ." Similarly Baldwin: "The patient died suddenly and without warning." Almost invariably the authors state that the operation progressed normally and the "color of the patient was good." Considerable cyanosis was reported only by Hahn and Davies. The respiration was restored for ten minutes in Baldwin's Case 11 following administration of artificial respiration for one hour. In all other cases failure of respiration was permanent. Death occurred in different stages of the operation with the exception of Baldwin's Case 2, who died a few minutes after the operation.

The relation of the failure of circulation to that of respiration varied in fatal cases. In McKean Downs' cases . . . "the heart continued to beat at first quite forcefully, then gradually faded out until it too stopped about one hour and a half after the breathing." Other authors reported simultaneous cessation of circulation and respiration. In Baldwin's Case 3, who was undergoing an abdominal operation, the circulation ceased instantaneously, the aorta becoming pulseless. Attempts at resuscitation with oxygen and in some cases with heart massage through the diaphragm were unavailing (Baldwin's Cases 9, 12, 13, Caine's Case 4, both of Hahn's cases).

(B) Cases with fatal outcome after hours, days or weeks. To this group belongs one case of Caine and our Cases 1, 2 and 3. The respiration ceased suddenly (Cases 1 and 3) or gradually (Case 2); but the failure of respiration was not permanent. It was reestablished after one and one-half minutes in Caine's case, after ten minutes in our Case 1 and after 40 minutes in our Cases 2 and 3. In none of these cases did respiration or circulation return to normal. The respiration was labored and 40 per minute in Case 1; it varied between 24 and 60 in Case 2; between 30 and 42 in Case 3, and between 30 and 56 in Caine's case. The pulse rate varied from 120 to 160 in Case 1; between 120 and 145 in Case 2; between 110 and 160 in Case 3, and 138 and 150 in Caine's case. The blood pressure in Cases 1 and 3 dropped slightly but soon returned to normal. There was a marked elevation of temperature in all instances: 108° (Case 1); 105° to 107° (Case 2); 106° (Case 3), and 102° to 104° (Caine). All reflexes were permanently abolished in Case 1. The reflexes were not reported in other cases. Convulsions, muscular twitchings, hypertonicity of extremities and trunk were present in all three cases. Cases 1 and 3 were comatose until death. There was a partial return of consciousness after nine hours in Caine's case and after 48 hours in Case 2. Death occurred after 17 hours (Caine), 60 hours (Case 1), 72 hours (Case 2) and 119 hours (Case 3).

In an unusual case reported by Caine, death occurred two months after anesthesia. A white woman of 58 was operated upon for chronic appendicitis and cholecystitis. Respiration and circulation ceased after 50 minutes of nitrous oxide-oxygen anesthesia. Oxygen and heart massage restored respiration and circulation one minute later. Consciousness and reflexes began to return after 45 minutes. Three hours later the patient was able to respond to questions and talked intelligently after six hours. She was apparently normal next morning but complained of seeing white balls (question of retrobulbar neuritis). Three days later the patient complained of impaired vision, but the eyegrounds were normal. A neurologist diagnosed a possible cerebral edema and considered the patient's condition to be very serious. A progressive mental deterioration developed and death occurred two months after the operation.

Group 2. Cases with incomplete recovery. To this group belongs a case reported by Caine and our Case 4. Caine's patient, a mulatto woman, aged 37, was operated upon for uterine fibroids. Respiration and pulse stopped

suddenly (time not given). Oxygen and heart massage restored the heart beat within two minutes and respiration five minutes later. Following anesthesia there was generalized paralysis, the patient was blind and unable to speak. Three years later the patient was blind, paralyzed and her speech was unintelligible. After six years she had some voluntary motion in her hands and arms and could distinguish colors and objects. Her intelligence was apparently good but the speech remained unintelligible.

Our Case 4 differs from the others in that there was no disturbance during the anesthesia, the clinical symptoms of brain damage appearing several hours after the operation. The clinical picture, however, is related to both the fatal and non-fatal cases but is less severe. She was semicomatose and greatly agitated. Convulsions, muscular twitchings, and disturbances of temperature, pulse and respiration were of the same type as in fatal cases. The paralysis was restricted to the left side and the eyesight was temporarily impaired (retrobulbar neuritis), resembling the picture described by Caine. The neurologic status varied during convalescence, tendon, reflexes and muscle tone showing frequent variations. The patient made a slow recovery and when seen five years later showed residual neurologic findings but was able to earn her living.

Discussion of Pathology.—In some of the fatal cases in which an autopsy was performed (McKean Downs, Hahn), the cause of death was not established and histologic examination of the brain was not reported. It is not likely that definite changes of the parenchyma could be demonstrated if death occurred instantaneously. Such changes can be expected only if the patient survived at least 24 to 36 hours following the anesthesia. From the neurologic findings in all three of our cases we can assume that death was due to the destruction of brain parenchyma. The two possible causes of this destruction which may be considered are: (1) asphyxia, (2) toxic effect of the gas.

The asphyxia might be produced in two ways: (*a*) anoxemia due to low oxygen content of the blood; (*b*) anoxemia due to collapse of the brain capillaries.

(*a*) The first possibility has been advocated by German authors (Flörcken[6], Lotheisen[7], Sudeck-Schmidt[8]) who advised the avoidance of cyanosis in the administration of nitrous oxide. This explanation is not entirely satisfactory. The significance of cyanosis is variously interpreted. According to Moore,[9] it is not necessarily a sign of oxygen deprivation and according to Raginsky and Bourne[10] a moderate degree of cyanosis cannot always be prevented. In most of the fatal cases no cyanosis was present and the color was good at all times (Case 2). There were no signs of anoxemia and the narcosis was not considered too deep. As a rule, the authors remark that both narcosis and operation progressed normally and that the fatal outcome occurred suddenly and without warning. It is not likely that signs of anoxemia were overlooked. Similarly anoxemia would not be expected to occur in 20 seconds (Davies). Anoxemia due to oxygen deprivation should

be promptly relieved by the administration of oxygen and is therefore not dangerous.

(*b*) Collapse of the capillaries associated with stasis and anoxemia as a responsible cause of degeneration of the parenchyma is to some extent supported by the histologic findings of Case 1, in which there were hyperemia and perivascular hemorrhages. But this change is too limited to produce a complete destruction of the cortex, and in Cases 2 and 3 it was not found. These conclusions are in agreement with the experimental observations by Gianotti and Vannotti[11] who studied the influence of nitrous oxide-oxygen anesthesia upon capillaries in rabbits. Under the influence of a mixture of 90 per cent nitrous oxide and 10 per cent oxygen, the entire capillary system contracted, the contraction being replaced by dilatation as soon as anesthesia was established. New capillaries appeared and the color of the underlying tissue became increasingly red. The arterioles were engorged, the speed of the blood current was reduced, the capillaries were markedly but unevenly dilated, occasionally forming aneurysms. The blood corpuscles were agglutinated and moved slowly and irregularly. Similar dilatation was also visible in the veins in which the blood flow was retarded. Perivascular and diffuse hemorrhages occurred. This condition remained without essential change even in prolonged anesthesia. Collapse of the blood vessels and complete stasis did not occur, and these manifestations could be promptly relieved by the administration of oxygen. The authors emphasized that these findings differed fundamentally from those produced by an asphyxiating mixture of nitrogen-oxygen which easily caused collapse of the vessels and stasis. They concluded therefore that the anesthetic effect of a nitrous oxide-oxygen mixture is not obtained by asphyxiation. The histologic picture suggests that the destruction of the brain is due to the toxic action of nitrous oxide on the parenchyma. A definite selective destruction is noted, the cortex and the basal ganglia being much more severely damaged than the brain stem and the cerebellum, resulting in a clinical picture of decortification. Destruction of this type is frequently a toxic manifestation and similar findings have been noted in cases of poisoning by excessive doses of pantapon, morphine and ergoapiol. Therefore, death may be due to extensive destruction of the cortex and basal ganglia rather than to involvement of the respiratory area *per se*.

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THE PHYSIOLOGY OF MASSIVE PULMONARY EMBOLISM *

AN EXPERIMENTAL STUDY OF THE CHANGES PRODUCED BY OBSTRUCTION TO THE FLOW OF BLOOD THROUGH THE PULMONARY ARTERY AND ITS LOBAR BRANCHES

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DESCRIPTIONS of the symptoms presented by patients with massive pulmonary embolism have usually been lacking in detail and clarity. This is probably due to the dramatic character of the event and to the usually rapid and fatal termination. Giertz and Crafoord,[1] however, have given an excellent account of the symptomatology in 27 cases. The symptoms observed in order of frequency were as follows: sudden onset of symptoms without any warning; high, soft, finally imperceptible pulse; marked pallor; unconsciousness; slight cyanosis; craving for air; dyspnea; altered respiration, superficial or deep; snatching or groaning; feeling of oppression, dread, anxiety, restlessness; pain or stitch in chest, usually over precordium; shock; cold perspiration; pulsations in veins of neck (the authors remark that this symptom is probably more frequent than was noted in the brief accounts available); waving arms about, wanting to get up. Violent vomiting, strabismus, dilated pupils, giddiness, and yawning also occasionally occur. They summarize their findings as follows: "In most instances it (massive pulmonary embolism) comes like a bolt from the blue with typical symptoms of which the most frequent are intensive pallor, loss of pulse and consciousness. Common symptoms are also oppression, craving for air and a mild cyanosis with a typical venous pulsation above the clavicles."

No adequate account of the physiologic changes underlying these varied symptoms has yet been given, although in a previous paper one of us[2] attempted to analyze the factors that lead to death when the main trunk of the pulmonary artery is occluded either completely or partially by a massive embolus. The complexity of the problem is increased because the obstruction to the flow of blood is usually not confined to a single point in the pulmonary arterial tree. The emboli are long and narrow and may not only produce a partial or complete occlusion of the main pulmonary artery but may extend into one or both main branches and obstruct a varying number of lobar branches.[3] There results a combination of partial occlusion of the main artery and a complete block of one or more of its lobar branches. The

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disturbance produced by partial occlusion of the pulmonary artery itself obviously differs from that produced by occlusion of certain branches of that artery and a difference in symptomatology results. Valuable information on this point could be obtained by an accurate correlation of the clinical symptoms with the location of the emboli found at autopsy. Unfortunately such correlation is rare.

Smellie,[4] however, has given a very good account of the symptoms presented by a patient with the obstruction to blood flow, incomplete but clearly confined to the main pulmonary artery. The patient, a boy ten years old, went to bed at 10 P.M. apparently perfectly well. At 1 A.M. he felt cold, and half an hour later vomited and complained of thirst. He vomited again at 3 A.M. At 5 A.M. he appeared to be asleep and breathing normally but looked pale. At 9 A.M. his general condition was the same. He did not reply when spoken to. At 10 A.M. he was breathing regularly but was very pale and could not be roused. He was taken to a hospital and died one-half hour later. At autopsy a large, firm, grayish-white clot was found in the right heart. It was firmly adherent in only one place, *i.e.*, the right auricular appendage. From this place of origin it extended through the right ventricle and terminated in each pulmonary artery just distal to the bifurcation. The thrombus was not adherent to the walls of the artery. There was an area of infarction about three inches in diameter in the lower lobe of the right lung. No other significant pathologic changes were found except some petechial hemorrhages beneath the epicardium. In this patient pallor and unconsciousness were the striking features, and except for the small area of infarction the obstruction was clearly confined to the main pulmonary artery.

Rochet,[5] in 1930, made a clinical distinction between two types of fatal pulmonary embolism, and characterized them as "syncopale" and "asphyxique." He gives the following descriptions of each type (translated by authors):

"I found a man propped up in bed, pale as a corpse and covered with cold sweat. The pupils were dilated, respirations were intermittent and tracheal râles were present. The pulse was imperceptible and the heart sounds were infrequent and muffled. There was no cyanosis. Death occurred a few minutes later. This is the 'syncopale' type of fatal pulmonary embolism.

"My second patient was a woman, age 35, upon whom I had operated for a large fibroadenoma of the breast. . . . The postoperative course was normal for two days. On the third day the patient while moving her bowels after an enema experienced a terrific pain in the back of the chest. It was 10 A.M. . . . Symptoms of asphyxia were predominant. Respirations were shallow and rather slow. The lips were black, in sharp contrast to the leaden pallor of the face. The hands and feet were deeply cyanotic. The pulse was thready and the rate could not be determined. Auscultation of the chest revealed no abnormalities. I ordered inhalations of oxygen and an

hypodermic injection of morphine to control the severe pain in the chest. I saw the patient an hour later. She had no more pain and was almost euphoric. The respiratory rhythm was normal but the cyanosis was as intense as before, and the flickering heart action persisted, despite the use of cardiac stimulants. At 5 P.M. her condition was the same or perhaps slightly better. Death occurred suddenly at 11 P.M. This is an example of the 'asphyxique' type of fatal pulmonary embolism."

Unfortunately autopsies were not performed on these cases. Judging by Smellie's case and our own experience, it seems likely that the "syncopale" type represents an obstruction of the main pulmonary artery. The clinical descriptions are decidedly similar. The "asphyxique" type on the other hand is better interpreted as an extensive block of the peripheral branches of the pulmonary artery. Evidence substantiating these clinical observations can best be obtained by separately producing both types of obstruction to blood flow in animals and recording the physiologic changes which follow. Some experimental work on this subject has already been performed.

In 1921 Underhill[6] noted the occurrence of anoxemia after ligating the left pulmonary artery in cats. The anoxemia was associated with some increase in pulmonary ventilation. Haggart and Walker,[7] in 1923, noted an average increase of 26 per cent in pulmonary ventilation following ligation of an artery to one lung. This was the result of an increase in tidal air and a slight increase in respiratory rate. Binger, Brow and Branch,[8] in 1924, produced multiple pulmonary emboli in animals by the intravenous injection of two or three hundred seeds, and noted the appearance of tachypnea and anoxemia. They concluded that the "tachypnea is due to anoxemia and can be stopped or prevented by oxygen inhalation. The anoxemia has been attributed to a change in the quantitative relation of blood flow to the vascular diffusion area in the lungs. The nature of this changed relationship is twofold: (*a*) an increased rate of flow through the capillaries, the flow being so rapid that the blood cannot assume its normal load of oxygen, (*b*) a compensatory dilatation in the capillaries which are crowded with corpuscles in columns so thick as to interfere with the normal inward diffusion of oxygen. Each of these defects in oxygen diffusion can be remedied by raising the alveolar oxygen tension." They did not attempt to determine quantitatively the diminution in the pulmonary vascular bed produced by the seed emboli.

The circulatory effects of partial occlusion of the main pulmonary artery have been studied by Gibbon, Hopkinson and Churchill.[9] In contrast to previous work on this subject it was found that a slow fall in systemic blood pressure could be produced by compression of the pulmonary artery. The decline in blood pressure is associated with a rise in venous pressure and a decrease in cardiac output. An illustration of the fall in blood pressure accompanying a massive pulmonary embolus has been given by Churchill[2] in a graphic chart of the blood pressure of a patient who finally succumbed after 17 hours. In the present investigation further studies upon the effects of compression of the main pulmonary artery are reported. In addition a vary-

ing number of branches of the pulmonary artery were occluded and the effects upon the cardiorespiratory system noted.

Our experiments have been divided into two groups. In Group A the blood pressure, the saturation of arterial blood with oxygen and the respiratory minute volume and tidal air were determined before and during partial occlusion of the main pulmonary artery. In Group B similar determinations were made before and during complete occlusion of a varying number of the lobar branches of the main pulmonary artery.

METHOD.—Cats anesthetized by the intraperitoneal injection of sodium barbital (0.45 g. per Kg. of body weight) were used in all experiments. The carotid arterial pressure was recorded on a kymograph in the usual manner with a mercury manometer. A cannula was introduced into the trachea and connected through a T-tube with inspiratory and expiratory Krogh valves. The expired air was collected by connecting the expiratory valve with a Benedict-Roth recording spirometer. Three minute collections of expired air were made to compute the respiratory minute volume. Samples of arterial blood were withdrawn under oil from a femoral artery and the oxygen content and capacity determined.

In Group A the pulmonary artery was compressed by a clamp capable of very fine adjustments.[10] The pulmonary artery was exposed in all but three experiments by removing a portion of the sternum, opening the pericardium, and making an airtight union between the opening in the pericardium and the opening in the wall of the chest.[11] With this preparation the pulmonary artery may be compressed and observations carried out in a normally breathing animal. In three experiments (Nos. 2, 3 and 4) the clamp was adjusted about the pulmonary artery through an intercostal incision and a small opening in the pericardium. The intercostal incision was then closed tightly around the shaft of the clamp. This preparation also permitted compression of the pulmonary artery in a normally breathing animal.

In Group B a varying number of lobar branches of the pulmonary artery were completely occluded by ligature. In all the experiments in this group the pericardium was sutured to the wall of the chest in the manner described by Drinker.[11] This gave access to the right and left main branches of the pulmonary artery in a naturally breathing animal. When the preparation was completed a record was made of the blood pressure, a sample of arterial blood was withdrawn for analysis and a record of the respiratory minute volume was made. A previously placed ligature was then tied about the right or left pulmonary artery completely occluding it. The observations were then repeated. When it was desired to shut off the blood supply to more than one lung, ligatures were tied about one or more lobar branches of the left pulmonary artery. The pericardium was then sutured to the wall of the chest. Occlusion of the right pulmonary artery then directed the entire output of the right heart through the unligated branches of the left pulmonary artery. Autopsies were always performed at the conclusion of the experiments to ensure that the vessels tied had remained completely occluded.

When it was desired to exclude the left middle lobe it was found to be technically simpler to ligate the pulmonary veins from this lobe instead of the artery. This produced the same fractional occlusion of the pulmonary circulatory bed that arterial occlusion would have accomplished. The known differences between the effects of vein and artery ligation in the lung were not considered significant to the problem at hand.

In order to estimate approximately how much of the total pulmonary vascular bed had been excluded from circulation by these ligations, the volume of pulmonary tissue represented by the right lung and the various lobes of the left lung was determined in ten normal cats. The trachea was clamped and the thoracic contents removed en masse. The lungs were then dissected free and the bronchi to the right lung and to the left upper, middle and lower lobes ligated. The right lung and the various left lobes were then separately immersed in water, the water displacement measured, and the percentage of the total lung volume calculated. The right lung varied between 54 and 66 per cent of the total lung volume, averaging 59.3 per cent. The left upper lobe varied between 7 and 9 per cent, averaging 7.4 per cent; the left upper and middle lobes together between 11 and 16 per cent, averaging 13.5 per cent; and the left lower lobe between 22 and 32 per cent, averaging 27.2 per cent. These averages were used in estimating the per cent of the pulmonary vascular bed excluded from the circulation by ligations of the branches of the pulmonary artery (Tables III and IV).

RESULTS.—Group A: Table I shows the systemic arterial blood pressure and the percentage saturation of arterial blood with oxygen before and dur-

TABLE I

BLOOD PRESSURE AND SATURATION OF ARTERIAL BLOOD WITH OXYGEN BEFORE AND DURING COMPRESSION OF THE MAIN PULMONARY ARTERY

			Before Partial Occlusion of the Pulmonary Artery			During Partial Occlusion of the Pulmonary Artery	
Experiment No.	Date	Weight of Cat	Systemic Blood Pressure	Saturation of Arterial Blood with Oxygen	Occlusion of Pulmonary Artery	Systemic Blood Pressure	Saturation of Arterial Blood with Oxygen
		Kg.	Mm. Hg.	Per Cent	Per Cent	Mm. Hg.	Per Cent
1	Oct. 15, 1930	2.64	125	90.2	undetermined	81	94.0
2	Oct. 22	2.57	103	89.8	undetermined	80	92.8
3	Oct. 27	3.00	140	94.0	undetermined	85	94.8
4	Oct. 29	2.84	83	94.8	undetermined	72	95.8
5	Oct. 31	2.85	126	91.7	88	90	93.6
6	Nov. 3	3.47	105	94.4	87	78	96.9
7	Nov. 5	3.55	78	92.0	61	62	92.8
8	Nov. 6	2.50	68	93.8	83	56	95.4

ing partial occlusion of the main pulmonary artery by the clamp. In Experiments 5 to 8, inclusive, the occlusion of the artery produced by the clamp was estimated in percentage of the original cross-sectional area.[9] As previously shown, occlusion of approximately 60 per cent of the cross-sectional area of the artery produces a fall in systemic blood pressure. In all these experiments the pulmonary artery was compressed until a definite drop in blood pressure occurred. Without exception the saturation of arterial blood with oxygen increased slightly, from 0.8 to 3.8 per cent, when the pulmonary artery was compressed.

In Table II the changes in respiratory minute volume, respiratory rate and tidal air with partial occlusion of the pulmonary artery are shown. When the occlusion was sufficient to produce a drop in blood pressure, there was an increase in the respiratory minute volume. The respiratory rate showed a variable change and the tidal air was consistently increased during compression of the pulmonary artery.

TABLE II

RESPIRATORY MINUTE VOLUME, RESPIRATORY RATE AND TIDAL AIR BEFORE AND DURING COMPRESSION OF THE MAIN PULMONARY ARTERY

Experiment No.	Date	Weight of Cat	Occlusion of Pulmonary Artery	Systemic Blood Pressure	Respiratory Minute Volume	Change in Respiratory Minute Volume	Respiratory Rate	Change in Respiratory Rate	Tidal Air	Change in Tidal Air
		Kg.	Per Cent	Mm.Hg.	Cc.	Per Cent	Per Min.	Per Cent	Cc.	Per Cent
9	Oct. 21, 1930	2.8	0	95	995		50		19.9	
			+	76	1,068	+ 7	44	−12	24.3	+22
			+ +	63	1,140	+15	45	−10	25.3	+27
10	Feb. 24, 1931	3.18	0	113	860		28		30.7	
(a)			84	65	1,089	+27	30	+ 7	36.3	+18
(b)	Feb. 24	3.18	0	99	1,026		28		36.6	
			61	98	1,026	0	23	−18	44.6	+22
(c)	Feb. 24	3.18	0	98	819		19		43.1	
			77	75	1,285	+57	20	+ 5	64.2	+49

Group B: The changes in blood pressure and arterial oxygen saturation following ligation of branches of the pulmonary artery are shown in Table III. Up to 67 per cent occlusion of the pulmonary vascular bed there is no significant change in the blood pressure. In Experiment 18 with an occlusion of 73 per cent there was a definite rise in blood pressure. With 86 per cent occlusion, *i.e.*, with the pulmonary circulation confined to the left upper

and middle lobes, there was a definite lowering of the blood pressure in Experiment 14, and a rise followed by a drop in Experiment 16.

TABLE III

BLOOD PRESSURE AND SATURATION OF ARTERIAL BLOOD WITH OXYGEN AFTER PARTIAL OCCLUSION OF THE PULMONARY VASCULAR BED

Experiment No.	Date	Weight of Cat	Pulmonary Tissue Receiving Output of Right Heart	Occlusion of Pulmonary Vascular Bed	Systemic Blood Pressure	Saturation of Arterial Blood with Oxygen
		Kg.		Per Cent	Mm. Hg.	Per Cent
11	Dec. 8, 1930		Both lungs	0	99	90.9
			Right lung	40.7	96	87.3
12	Dec. 10	2.72	Both lungs	0	86	
			Left lung	59.3	92	
13	Dec. 12	2.6	Both lungs	0	81	84.7
			Left lung	59.3	84	87.1
14	Dec. 13	2.58	Right lung and left upper and middle lobes	27.2	120	
			Left upper and middle lobes	86.5	95	
15	Dec. 19	3.04	Right lung and left lower and middle lobes	7.4	96	92.1
			Left lower and middle lobes	66.7	100	89.4
16	Jan. 5, 1931	5.11	Right lung and left upper and middle lobes	27.2	136	87.9
			Left upper and middle lobes	86.5	168–92	32.6
17	Feb. 10	3.81	Right lung and left lower lobe	13.5		92.0
			Left lower lobe	72.8		86.3
18	Feb. 12	4.15	Right lung and left lower lobe	13.5	89	
			Left lower lobe	72.8	123	
19	Feb. 18	5.05	Right lung and left upper and middle lobes	27.2		91.6
			Left upper and middle lobes	86.5		24.7

With one exception (No. 13), there was a definite drop in the arterial oxygen saturation following occlusion of 41 to 86 per cent of the pulmonary vascular bed. In Experiment 13 there was an abnormally low arterial oxygen saturation in the control period *before* ligating the right pulmonary artery. This was the only experiment in which an attempt was made to withdraw arterial blood from the left ventricle by direct puncture. The attempt was unsuccessful and immediately afterward an arterial sample with low oxygen saturation was obtained from a femoral artery. In two experiments (Nos. 16 and 19) there was a profound drop in the saturation of

arterial blood with oxygen following 86.5 per cent occlusion of the pulmonary vascular bed. In Experiment 16 the animal was killed by accidental inhalation of water three and one-half minutes after confining the pulmonary circulation to the left upper and middle lobes and two minutes after the blood sample for oxygen determination had been withdrawn from the femoral artery. In Experiment 19 the animal was revived by the inhalation of oxygen following which the arterial oxygen saturation rose from 24.7 to 68.5 per cent. In Experiment 14, with a similar occlusion of 86.5 per cent, the animal died six minutes after ligation of the right pulmonary artery. No samples of blood were withdrawn for analysis in this experiment.

The respiratory minute volume, respiratory rate and tidal air with varying degrees of occlusion of the pulmonary vascular tree are shown in Table IV. There was always an increase in respiratory minute volume; a variable change, generally an increase, in respiratory rate; and a consistent increase in tidal air.

TABLE IV

RESPIRATORY MINUTE VOLUME, RESPIRATORY RATE AND TIDAL AIR WITH PARTIAL OCCLUSION OF THE PULMONARY VASCULAR BED

Experiment No.	Date	Weight of Cat	Pulmonary Tissue Receiving Output of Right Heart	Occlusion of Pulmonary Vascular Bed	Respiratory Minute Volume	Change in Respiratory Minute Volume	Respiratory Rate	Change in Respiratory Rate	Tidal Air	Change in Tidal Air
		Kg.		Per Cent	Cc.	Per Cent	Per Min.	Per Cent	Cc.	Per Cent
12	Dec. 10, 1930	2.72	Both lungs	0	387		15.5		25.0	
			Left lung	59.3	577	+49	18.5	+19	31.2	+20
13	Dec. 12	2.6	Both lungs	0	466		30.5		15.3	
			Left lung	59.3	487	+ 5	30.0	− 2	16.2	+ 6
14	Dec. 13	2.58	Right lung and left upper and middle lobes	27.2	887		35.5		25.0	
			Left upper and middle lobes	86.5	1,333	+53	36.5	+ 3	36.5	+46
15	Dec. 19	3.04	Right lung and left lower and middle lobes	7.4	621		23.5		26.4	
			Left lower and middle lobes	66.7	987	+59	31.0	+32	31.8	+20
16	Jan. 5, 1931	5.11	Right lung and left upper and middle lobes	27.2	1,529		23.5		65.0	
			Left upper and middle lobes	86.5	3,448	+125	30.0	+28	114.9	+77
18	Feb. 12	4.15	Right lung and left lower lobe	13.5	446		17.5		25.5	
			Left lower lobe	72.8	833	+87	19.5	+11	42.7	+67

DISCUSSION.—The lowered blood pressure with varying degrees of occlusion of the pulmonary artery is shown in Table I. An occlusion of approximately 60 per cent produces a significant lowering of the systemic blood pressure. The lowered blood pressure is at least in part due to the diminished cardiac output resulting from constriction of the pulmonary artery.[9] On the other hand, there is no lowering of the blood pressure with occlusion of up to 86 per cent of the peripheral pulmonary vascular bed, as can be seen in Table III. Indeed, in one instance (No. 18) there was a definite rise in pressure. The probable explanation for this difference lies in the fact that a small fraction of the pulmonary vascular bed can accommodate the entire

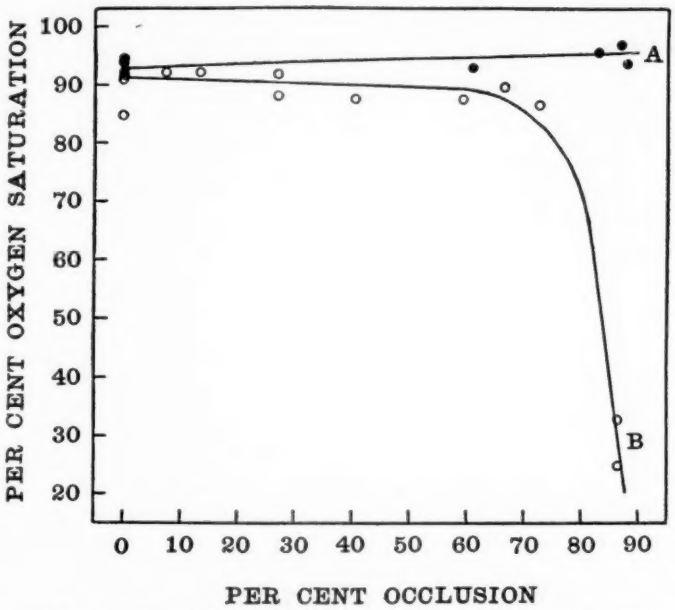

CHART 1.—Graphic illustration of the difference in arterial oxygen saturation with occlusion of the pulmonary artery (A) and with occlusion of its lobar branches (B). The solid dots and the straight line (A) represent the percentage saturation of arterial blood with oxygen before and during partial occlusion of the pulmonary artery. The circles and the curved line (B) represent the percentage saturation of arterial blood with oxygen before and during partial occlusion of the pulmonary vascular bed.

output of the right heart. There is already some evidence that an increased blood flow is associated with a lowering of resistance in the pulmonary vessels.[12]

The slight increase in the percentage saturation of arterial blood with oxygen observed with compression of the main pulmonary artery (Table I and Chart 1) may be due to a slower movement of blood throughout the whole pulmonary capillary tree, because of the diminished output of the right heart. A more complete saturation of the blood with oxygen would tend to result from the slower passage of blood through the pulmonary capillaries. On the other hand, when the output of the right heart is confined to the blood vessels of one lung, or a part of one lung, the opposite effect obtains

(Table III and Chart 1). A 40 to 75 per cent occlusion of the pulmonary vascular bed resulted, with one exception, in a slight but significant reduction in the saturation of arterial blood with oxygen. Eighty-five per cent occlusion produced marked lowering in the oxygen content, and proved fatal without the inhalation of oxygen in two instances. In the third instance the animal was accidentally killed a few minutes after the occlusion was effected. The explanation offered by Binger[8] may well apply here. The pulmonary vessels are congested and the blood flow is rapid. This results in an impaired diffusion of oxygen into the blood and a consequent lowering of the per cent saturation.

No significant differences in the respiratory changes resulting from partial occlusion of the pulmonary artery and partial occlusion of the pulmonary vascular tree were observed in the few experiments performed. With compression of the pulmonary artery sufficient to cause a drop in blood pressure there was an increase in total pulmonary ventilation, a variable change in respiratory rate and a consistent increase in tidal air (Table II). That these changes are not due to a decrease in oxygen saturation of arterial blood is evident (Table I). They might be due to the reduction in cardiac output[9] occasioning an accumulation of carbon dioxide or possibly a lack of oxygen in the respiratory center. A similar increase in total pulmonary ventilation and tidal air occurs with partial occlusion of the pulmonary vascular bed (Table IV). There is also generally an increase in the respiratory rate. These changes are due to the decreased oxygen saturation of arterial blood and can be prevented by oxygen inhalation.[8] This was illustrated in Experiment 19 (not included in Table IV) in which a rapidly developing respiratory and circulatory failure following 86 per cent occlusion of the pulmonary vascular bed was overcome by administering oxygen. The percentage saturation of arterial blood which had fallen to 24.7 was raised to 68.5 per cent. Prior to the occlusion, the respiratory minute volume was 1,492 cc., the respiratory rate 28 per minute, and the tidal air 53.3 cc. After the occlusion and with the administration of oxygen, the figures were 1,264, 30 and 41.8 respectively.

SUMMARY.—Occlusion of more than 60 per cent of the main pulmonary artery results in a reduction of cardiac output, a fall in systemic blood pressure, an elevation of venous pressure, a slight increase in the saturation of arterial blood with oxygen, an increase in total pulmonary ventilation and tidal air and a variable change in respiratory rate.

With an occlusion of 40 to 73 per cent of the peripheral vascular bed of the lungs, the systemic blood pressure remains constant or is elevated, the saturation of arterial blood with oxygen is decreased and there is an increase in total pulmonary ventilation and tidal air with a variable change in the respiratory rate.

When 86 per cent of the pulmonary vascular bed is occluded, there is a fall in systemic blood pressure and a profound decrease in the saturation of

arterial blood with oxygen resulting in death. Inhalation of oxygen increases the oxygen saturation of the blood and maintains life.

These experimental findings aid in the interpretation of certain clinical observations in patients with pulmonary embolism.

CONCLUSIONS

These experimental observations point the way to a clearer understanding of the symptoms of pallor, cyanosis, air hunger, *etc.*, which have been observed in patients with massive pulmonary embolism. The "syncopale" type observed by Rochet would correspond to an experimental occlusion of the main pulmonary artery. The "asphyxique" type corresponds to the experimental occlusion of the larger branches of the pulmonary artery. From the experimental evidence the following presumptions seem justified. Where symptoms of pallor and lowered blood pressure predominate in a patient suffering with pulmonary embolism, the embolus produces the greatest degree of obstruction in the pulmonary artery at or above the point of bifurcation. Where cyanosis is a prominent feature and the lowering of the blood pressure is slight or absent, the embolus blocks at least one main branch of the pulmonary artery, and possibly some of the smaller branches of the other. More exact conclusions might be reached by determining the percentage saturation of arterial blood with oxygen. Finally, oxygen inhalations may be a life saving measure where there is marked diminution in the pulmonary vascular bed, and hence should certainly be given in all cases with the slightest evidence of cyanosis.

The respiratory changes occur in both types of obstruction and although perhaps more marked with occlusion of the peripheral vascular bed the difference is not great enough to be significant. The increased depth of respiration, however, remains an important diagnostic feature in all cases of massive pulmonary embolism. Elevation of the venous pressure regularly occurs with the obstruction of the main pulmonary artery.[9] On two occasions the venous pressure was measured in the peripheral type of obstruction. In one instance there was an increase of 10 Mm. water pressure with 86 per cent occlusion, and in the other no change was observed with 73 per cent occlusion.

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PULMONARY EMBOLECTOMY

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NO SUCCESSFUL case of operative intervention in massive pulmonary embolism has been reported in this country, and but few unsuccessful attempts are recorded.[1, 2, 3, 4, 5] The European surgeons who have had the most experience with the Trendelenburg operation, some of them successful, have felt that the procedure should be more widely practiced and have urged that even the unsuccessful cases be reported in order that the diagnostic and technical difficulties may be clarified.[6, 7, 8, 9] With this in mind, it seems appropriate to describe a recent unsuccessful embolectomy, particularly since this experience has demonstrated to us the feasibility of the procedure and has given us reason to hope that further efforts may prove successful.

CASE REPORT

A man, age 59, was convalescent following a suprapubic prostatectomy when, on the tenth postoperative day, he experienced a sudden attack of dyspnea with moderate cyanosis. This attack lasted only a few minutes and the patient was comfortable until 24 hours later when, while eating supper, a second more severe seizure occurred. The patient was seen at 4:55 P.M. two or three minutes after the onset of symptoms. He was sitting up in bed with a slightly cyanotic pallor and was gasping for breath. His pulse was very rapid and weak. He was rushed to the operating room thrashing about so wildly that it seemed he would throw himself out of bed. Just as he was being wheeled into the amphitheater, however, he became motionless, respirations ceased, and the heart sounds could not be heard at the apex. The chest was quickly shaved and the operation was begun without anesthesia. The Trendelenburg armamentarium, which is kept constantly in readiness, provided sterile gown, gloves, and instruments. The patient at this time, about eight minutes from the onset of symptoms, was clinically dead.

The second, third and fourth costal cartilages were resected through a straight incision along the left border of the sternum, and the outer third of the sternum was removed with large rongeurs. There was no bleeding. The pericardium was opened with some difficulty because of the enormously distended right ventricle bulging up beneath. The heart was motionless. The Trendelenburg sound was passed behind the aorta and pulmonary artery, the rubber tourniquet was drawn into place and the pulmonary artery was opened just distal to the valve by a longitudinal incision 2 cm. in length. A firm clot 30 cm. long and 2 cm. wide at the center, tapering at either end, was removed from the right branch. No clot was found in the left branch. The incision in the artery was closed with the Trendelenburg clip and the vessels allowed to drop back into place. About five minutes had elapsed since the cessation of respiration. The heart was massaged manually for a few moments without result. Five cc. of adrenalin were injected into the heart and further massage resulted in a faint irregular pulsation which gradually increased in amplitude, becoming a regular full beat. During this period the heart was irrigated with warm saline solution and artificial respiration was carried on through a metal airway. A small rent which had been torn in the pleura was plugged with moist cotton. After about ten minutes, spontaneous respiration was resumed which became regular and deep. The patient's color returned to normal, he began to perspire

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and there were slight movements of the extremities. He remained in this favorable condition for some 30 minutes, when, just as preparations were being made to suture the artery, the clip slipped off, apparently due to the vigorous motion of the heart. It was replaced with some difficulty in the bloody field by reapplying traction on the tourniquet. On release the heart dilated rapidly and stopped beating, assuming the appearance it had shown on opening the pericardium. Inspection showed that the clip had been placed so as to almost occlude the artery. This was readjusted but measures to restore the heart action were now without avail. Permission for autopsy was not obtained.

The Trendelenburg operation has been undertaken at the Peter Bent Brigham Hospital four times during the past three years. During this period the diagnosis of massive pulmonary embolism has been made in 12 patients, all of whom died. In several of these cases an autopsy was not made, but the clinical picture was quite typical. In three of the four operated cases the diagnosis was confirmed at operation. In the fourth case no clot was recovered so the diagnosis may have been in error, but as no autopsy was made, this could not be established. In six cases death had occurred by the time the House Officer had reached the bedside. The patient described above died eight minutes after the onset of symptoms, and was revived temporarily by embolectomy. Two patients survived the embolism by 15 minutes but operation was not attempted. Two patients survived for 20 minutes. Both of these were subjected to embolectomy, by Dr. R. Zollinger and by Dr. E. C. Cutler, the first just after death, and the second just before death. In neither of them could the heart action be restored after extraction of the clots. In the second case the aorta was opened by mistake and had to be sutured before proceeding with the embolectomy.

In the fourth case, operated upon by Dr. E. C. Cutler, the pulmonary artery was explored just after death but no clot could be found. Although autopsy was refused, it seems possible that there was a friable embolus which had been scattered into the smaller branches of the pulmonary artery out of reach of the forceps or suction. The clinical circumstances were typical of pulmonary embolism.

CASE REPORT

A young robust woman of 34 had, 18 months previously, suffered a very definite though non-fatal pulmonary embolism following the excision of postphlebitic ulcers of the legs. The ulcers had recurred and a second excision and grafting had been performed. Three weeks later a superficial phlebitis of the left thigh appeared and, at about the same time, a pleuritic pain in the left chest. Four days later there was a sudden attack of severe dyspnea and cyanosis followed shortly by pallor and loss of consciousness. She was seen within a few minutes by the House Officer who noted that the neck veins were distended. Five minutes later, when the operation was begun, respiration and heart action had ceased.

The difficulty of avoiding injury to the pleura has been stressed by all of the surgeons who have attempted embolectomy. The operative approach described by Meyer,[10] minimizes this hazard. An opening in the pleura, however, does not necessarily mean failure for a small defect can be effectively controlled with moist cotton. Nyström[6] injured the pleura in six cases,

including his three operative recoveries, out of a total of ten reported operations. The pleura was likewise injured in the patients, reported by Westerborn[7] and by Petterson,[9] who survived operation by 16 and 3 hours, respectively. The possibility of mistaking the aorta for the pulmonary artery must be kept in mind, especially in the exceptional instance where the right ventricle and pulmonary artery are not dilated. This error was made in the first case of Nyström's series and was likewise made in one of our cases. Both of these errors could have been prevented by sufficient practice on the cadaver. A third technical error was demonstrated in the case described in this paper. The clip was allowed to remain in place long enough to be gradually worked loose by the vigorous heart beat. It would seem wiser to have proceeded with the arterial suture as soon as the heart action had been restored.

Much has been written about the clinical diagnosis of massive pulmonary embolism. We have been impressed by the fact that the clinical diagnosis is only rarely mistaken. It must be admitted, however, that errors in diagnosis may occur and this fact added to the consideration that an occasional case of massive pulmonary embolism recovers spontaneously leads one to the conclusion that operative intervention should be undertaken only when the patient is moribund.

There has been some difference of opinion as to whether the operation should be undertaken only by senior surgeons or whether it should be entrusted to a member of the resident hospital staff. In order to determine the operative possibilities in this regard a survey was made of all the proven cases of death from massive pulmonary embolism in the Peter Bent Brigham Hospital during the past 23 years. There were 92 cases in all. In 33 operation would have been out of the question because the patient was found dead or else died shortly after the initial symptoms (Table I).

TABLE I

Found dead in bed	5 cases
Died immediately during attack	9 cases
Lived five minutes or less after onset	19 cases

In 36 cases the patient survived for ten minutes to one hour (Table II).

TABLE II

Survived after onset for 10 minutes	8 cases
Survived after onset for 15 minutes	8 cases
Survived after onset for 20 minutes	11 cases
Survived after onset for 30 minutes	3 cases
Survived after onset for 45 minutes	5 cases
Survived after onset for 60 minutes	1 case

In this group it is reasonable to assume that successful surgical intervention would have to have been undertaken by someone in the hospital at the time of the accident. In only 12 of these cases, however, did this occur between the hours of 9 A.M. and 5 P.M., when a senior surgeon would have been available.

In 15 cases there was a survival period of one to 12 hours (Table III).

TABLE III

Survived after onset for 1 hour and 15 minutes..	3 cases
Survived after onset for 1 hour and 30 minutes..	3 cases
Survived after onset for 2 hours.....	4 cases
Survived after onset for 4 hours.....	2 cases
Survived after onset for 8 hours.....	2 cases
Survived after onset for 12 hours.....	1 case

In the group which comprise Table III there would have been sufficient time to call the senior surgeon. In general, however, these patients are not in the most favorable condition for operation, as the heart is likely to become too exhausted to be revived, or, as Churchill[4] has pointed out, a state of peripheral circulatory collapse may develop which probably would make recovery impossible. Moreover, secondary thromboses may occur within the pulmonary arborization.

In the final group of eight cases, the survival period was from one to five days. Here the likelihood of extensive thrombosis is so great that successful surgical intervention would seem out of the question.

CONCLUSIONS

The operation of pulmonary embolectomy must be undertaken by a member of the resident staff, if the procedure is to be ventured in the majority of the favorable cases.

It is believed that the clinical picture in these fatal cases is sufficiently clear cut that mistakes in diagnosis will be very few and that where the diagnosis is in error little harm can accrue since the patients are obviously moribund.

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ACUTE PERFORATION OF GASTROJEJUNAL ULCER*

REPORT OF TEN NEW CASES AND A REVIEW OF NINETY-THREE COLLECTED CASES

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THE term gastrojejunal ulcer is used in this paper to include all secondary ulcers situated at or adjacent to anastomoses between the stomach and jejunum irrespective of their gastric, marginal or jejunal location. The qualifying term acute perforation sometimes is used loosely in the literature to include subacute perforation, gastrojejunocolic fistula or fistula through the abdominal wall. It is restricted here to the use ordinarily made of it in this country with reference to perforation of peptic ulcer which corresponds to the German expression acute perforation into the free peritoneal cavity. Whereas ulcers which occur in the region of gastrojejunal anastomoses seem to possess the etiologic and pathologic features of primary peptic ulcer, they are to be distinguished from the latter by virtue of their occurrence in a small but definite proportion of cases as a sequel to surgical anastomosis. Synonyms include anastomotic ulcer, marginal ulcer, secondary jejunal ulcer, ulcus pepticum jejuni and peptic jejunal ulcer. Gastrojejunal ulcer is to be differentiated also from the rarely occurring primary jejunal ulcer.

Eighteen years after Woelfler[99] (1881) performed the first successful gastrojejunostomy, Braun[6] (1899) reported the first case of gastrojejunal ulcer. A point of interest here is that the first case of gastrojejunal ulcer to be reported was one in which acute perforation took place. Goepel[25] reported the first successful suture of acute perforation of gastrojejunal ulcer in 1902. In one of the earliest comprehensive papers on the subject, Paterson,[65] in 1909, originated the term gastrojejunal ulcer. At that time, ten years after the first case was reported, he stated that there were 52 authenticated cases of gastrojejunal ulcer on record.

ETIOLOGY.—*Incidence.*—Because it is known that many of the observed cases have not been reported, it is readily admitted by those who have written on this topic, that it is not possible to determine the frequency of occurrence of gastrojejunal ulcer. There are numerous casual references in the literature to cases of acute perforation of gastrojejunal ulcer. It is doubly difficult, therefore, if not impossible, to determine the incidence of this complication of gastrojejunal ulcer. Published estimates of the occurrence of gastrojejunal ulcer vary from 1.4 (Walton[95]) to 34 (Lewisohn[50]) per cent. In 129 cases of gastrojejunal ulcer collected by Lieblein,[53] there were 30 cases of acute perforation, or an incidence of 23 per cent. If we accept Sokolov's[82] and Lahey and Jordan's[48] estimates that 1,000 cases of

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gastrojejunal ulcer have been reported in the world literature, our search of the literature, which yielded 93 more or less complete case reports, would indicate an incidence of acute perforation in gastrojejunal ulcer of less than 1 per cent. Singer and Meyer[81] point out that there is a relatively large group of unrecognized cases of free perforation which, if added to the known cases, would increase the percentage of incidence materially.

Contributory Etiologic Factors.—Until the problem of the etiology of primary peptic ulcer is solved, it is not likely that the cause of secondary gastrojejunal ulcer will be found. Some of the factors contributory to the production of gastrojejunal ulcer, which seem to have a bearing on acute perforation, will be briefly reviewed.

Despite our lack of knowledge regarding the direct or ultimate cause of gastrojejunal ulcer, two very important facts stand out. One is the peculiar susceptibility of the jejunum (the tissue susceptibility factor of Ochsner and his co-workers[63]) to the influence of gastric juice. The other is the marked tendency of gastrojejunal ulcer to penetrate or perforate. The latter is attested to by the high frequency of subacute perforation of gastrojejunal ulcer and by the frequent occurrence of gastrojejunocolic fistula.

Many of the factors which seem to contribute to the incidence of uncomplicated gastrojejunal ulcer also appear to contribute to the frequency of complicating acute perforation of gastrojejunal ulcer. Factors on the part of the patient are constitutional predisposition (Ochsner, *et al.*[63]), early age, male sex, high concentration of free hydrochloric acid, coexisting infection, and failure to follow postoperative medical management. Technical considerations which should be avoided by the surgeon are anterior gastrojejunostomy, artificial occlusion of the pylorus (von Eiselsberg pyloric exclusion), entero-anastomosis (Braun's or Roux's en Y), the use of the Murphy button and non-absorbable sutures. Gastrojejunal ulcer and in turn acute perforation has been reported but it is rare following resection of the stomach for malignant disease. It appears, therefore, that the same factors which contribute to the production of benign peptic ulcer seem to be operative in the formation of secondary ulcer.

The most common site of gastrojejunal ulcer is in the efferent limb of the jejunum.

Pathology.—The pathogenesis of acute perforation of gastrojejunal ulcer is apparently the same as that of acute perforation of primary peptic ulcer except that the jejunal mucosa is more susceptible than that of the stomach or duodenum to the proteolytic effects of gastric juice.

The essential gross and microscopic pathology is the same as that of primary peptic ulcer. The ulcer which perforates acutely usually is single but may be multiple. Except in gastrojejunocolic fistula, the tendency to heal is less common than in gastric or duodenal ulcer. There is a marked tendency toward recurrence manifest in some perforating gastrojejunal ulcers. An outstanding feature, and the one which chiefly concerns us here, is this tendency to penetrate and involve other tissues. Based on this characteristic,

gastrojejunal ulcers may be divided into three groups: (1) Non-penetrating ulcer which, while it may erode a blood vessel or obstruct the stoma of the anastomosis, does not extend beyond the wall of the stomach or jejunum. (2) Chronic penetrating ulcer, which may form an indurated mass involving the transverse mesocolon, the colon, the omentum or the anterior abdominal wall with or without abscess formation, or which may penetrate into the colon or through the abdominal wall with fistula formation. (3) Acute perforating ulcer, which allows the escape of gastric or jejunal content into the free peritoneal cavity. This tendency to acute perforation of ulcer is so marked in some patients that in one group acute perforation of gastric or duodenal ulcer is found to have preceded the performance of gastrojejunostomy whereas in a second group multiple successive perforations of gastrojejunal ulcer have taken place.

Symptoms and Diagnosis.—Acute perforation of gastrojejunal ulcer may or may not be preceded by suggestive prodromal symptoms. When premonitory symptoms are present, they usually consist of recurring dyspepsia appearing from a few days to as long as 25 years after operation. The dyspepsia often is refractory to the usual alkali and dietary management. In gastrojejunal ulcer the pain is usually on the left of the midline and at or below the level of the umbilicus.

In acute perforation of gastrojejunal ulcer as in peptic ulcer the pain is usually sudden and severe. The subjective symptoms are so characteristic that in several recorded instances patients who have experienced previous perforations have made their own diagnosis. On examination, the greatest tenderness and rigidity may be in the left lower quadrant of the abdomen because the fluid which escapes through the perforation passes downward on the left of the vertebral column. The late symptoms and signs are those of perforative peritonitis.

In those patients in whom the evidence points to acute perforative peritonitis, the diagnosis of acute perforation of gastrojejunal ulcer is made chiefly on the history of the patient's having had a gastrojejunostomy performed. The most difficult but least important condition to be differentiated is perforation of recurrent gastric or duodenal ulcer, the primary and confirmatory signs of which, as recently shown by one of us (H.L.T.[91]), are characteristic in definite proportions of cases. In these latter conditions the recurrent symptoms more nearly simulate the original ulcer distress.

Prognosis.—The prognosis in acute perforation of gastrojejunal ulcer, treated or untreated, is grave. Most of the untreated cases are fatal. Wright[102] reported a mortality of 23 per cent in cases treated by simple suture with 10.4 per cent of good results and 31 per cent of which required further operation.

Treatment.—The prophylactic treatment of acute perforation of gastrojejunal ulcer begins with the treatment of gastric or duodenal ulcer, the principles of which are well known. Most students of gastrojejunal ulcer

are agreed that gastrojejunostomy should be restricted to cases in which pyloric obstruction is present and in which the acid values are low.

Except in late cases wherein progressive peritonitis has supervened, the active treatment of acute perforation of gastrojejunal ulcer is surgical. In late diffuse peritonitis the injudicious application of surgery may be not only harmful but fatal. With respect to treatment in early cases, it may be said that there are two schools of thought. The adherents to one school maintain that simple suture is safest and therefore sufficient for the primary operation and can be followed by medical management and by radical surgery later if required. The members of the second school believe that in selected cases curative measures are warranted. If the duodenal ulcer is healed and the pylorus is patent, the gastrojejunostomy may be taken down and normal continuity restored. If active peptic ulcer is present, pyloroplasty, gastroduodenostomy to the first or second portion of the duodenum or Pólya or Billroth partial gastrectomy may be employed.

It is hardly necessary to emphasize that adequate medical management, including elimination of infection, should be employed postoperatively when either primary or secondary ulcer is present.

REVIEW OF CASES REPORTED IN THE LITERATURE

Our review of the literature disclosed four interesting facts. The first is that relatively few exhaustive studies of this subject have been made. The most notable ones in English are those made by Paterson,[65] Massie,[56] and Singer and Meyer.[81] The second interesting fact is that only 93 authenticated case reports of acute perforation of gastrojejunal ulcer could be found. Singer and Meyer[81] state that the publication of Steden's[84] article in 1931 brought the total number of cases reported in the German literature to 20. In the general literature on gastrojejunal ulcer there are a number of casual references to cases of acute perforation into the free peritoneal cavity. Except from the standpoint of numbers these cases have little significance because they cannot in any way be considered as case reports useful for study. For example, Wright[102] mentioned that 51 cases had been reported to him by the Fellows of the Association of Surgeons. Sokolov[82] made a similar collection of 12 cases from among Russian surgeons. Hurst and Stewart[37] referred to eight cases and Graham and Lewis[26] to one case. The third interesting fact is that a number of cases reported as acute in reality have been subacute peforations or perforations into the colon or through the anterior abdominal wall. Singer and Meyer[81] credit Fromme,[24] Florcken and Steden,[22] McKelvey,[58] Recht[71] and Urrutia[94] with a total of ten cases whereas we accept only four of them as cases of acute perforation into the free peritoneal cavity. Moreover, personal communications indicate that through error Lewisohn[51, 52] mentioned one case and that Storey[88, 89] included a case of subacute perforation and a case of primary perforation of the jejunum in his incidental report of five cases of acute perforation of gastrojejunal ulcer. The fourth point of interest is that there is a good

deal of confusion regarding certain cases. As an illustration, Mikulicz is credited as having reported five cases. As early as 1909, Paterson[65, 66] classified three of these as doubtful and our study of the literature has disclosed that neither of the other two is a typical acute perforation of secondary ulceration at the gastrojejunal anastomosis. In 1899 and 1900, Kausch[44] reported from Mikulicz clinic a case of chronic perforating gastrojejunal ulcer which ultimately penetrated the anterior abdominal wall. In the Handbook of Surgery in 1903, Mikulicz and Kausch[60] made a parenthetic reference to a case wherein perforation of an acute gastrojejunal ulcer occurred in an infant soon after operation for hypertrophic stenosis of the pyloris. Freund,[23] in 1903, reported the same case from the standpoint of hypertrophic stenosis without reference to the gastrojejunal ulcer. Mikulicz,[59] also in 1903, reported briefly the same case to the American Surgical Association. Such instances of multiple reports of the same case have served to confuse rather than clarify the situation. In view of the foregoing facts and the fact that authenticated cases of acute perforation of gastrojejunal ulcer now number over 100, it seemed promising to combine these cases for a comprehensive study of a problem which is clinically distinct from that presented by primary peptic ulcer in any of its complicated forms or by gastrojejunal ulcer in any of the other forms in which it is seen to exist.

This study is made on a total of 103 cases, 93 of which were collected from the original case reports in the literature. It is our privilege to add ten new cases which are reported here for the first time. The latter group is composed of five cases of our own and five which were generously contributed to this study. For two of the latter we are indebted to Hinton[34] and for three to Storey[89] (Table VI).

Age and Sex.—Among the 92 cases in which the age was recorded there were cases in every decade of life from the first through the seventh. The single case in the first decade was in a child of five months. In the seventh decade there were two cases. Thirty-six per cent of the cases occurred in the fourth decade and 49 per cent in the third and fifth decades combined. Among the 99 cases in which the sex was recorded, 90 per cent occurred in males. Acute perforation of peptic ulcer therefore is predominantly a disease of the male sex and of the ages from 21 to 50.

Primary Pathology.—The primary pathologic condition for which gastrojejunostomy was performed was recorded in 89 cases (Table I). Duodenal ulcer was present in 43 per cent, gastric ulcer in 30 per cent. Pyloric stenosis was recorded as the primary pathology is 20 per cent of the cases. Carcinoma of the stomach, gastroptosis, gastric erosions, congenital hypertrophic stenosis of the pylorus, gastric and duodenal perforations and "no pathology found," each were reported in one case. It is probable that peptic ulcer was present as the cause of stenosis in many of the cases in the pyloric stenosis group.

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TABLE I

PRIMARY PATHOLOGY

	Cases	
	Number	Per Cent
Duodenal ulcer	38	42.6
Gastric ulcer	27	30.3
Pyloric stenosis	18	20.2
Carcinoma of stomach	1	1.1
Gastroptosis	1	1.1
Gastric erosions	1	1.1
Congenital hypertrophic stenosis of pylorus	1	1.1
Gastric and duodenal perforations	1	1.1
No pathology found	1	1.1
Totals	89	99.7

COMPLICATIONS OF PRIMARY PATHOLOGY

	Cases	
	Number	Per Cent
Acute perforation of peptic ulcer	24	27
Peptic ulcer with obstruction	20	22
Peptic ulcer with hemorrhage	11	12
Peptic ulcer with chronic perforation of pancreas	2	2
Totals	57	63

TABLE I—Showing the uncomplicated and complicated primary pathologic conditions for which gastrojejunostomy was performed, to be followed later by acute perforation of a gastrojejunal ulcer. Peptic (gastric and duodenal) ulcer constituted the primary pathology in 72.9 per cent of cases. Acute perforation was the most common complication of peptic ulcer to be followed by acute perforation of gastrojejunal ulcer.

One of the four common complications of peptic ulcer was present in 63 per cent of the 89 cases in which primary pathology was recorded (Table I). Acute perforation of peptic ulcer was present in 27 per cent, peptic ulcer with obstruction in 22 per cent, hemorrhage in 12 per cent, and chronic perforation into the pancreas in 2 per cent of cases. It should be noted that the most common complication of primary peptic ulcer was acute perforation. Obstruction (exclusive of pyloric stenosis) was the next most common complication.

ORIGINAL OPERATION.—The form of the original operation was recorded in all of the 103 cases (Table II). Among these gastrojejunostomy was used in 92 cases, the posterior form in 45 and the anterior in 40 cases. Pyloric resection was utilized in 11 cases with the Billroth II and Pólya types each having been applied in four instances. Operative procedures used in combination with the above in 24 cases were entero-anastomoses, which were used in 16 cases, and pyloric exclusion utilized in five cases,

excision of ulcer and the Roux en Y operation which were used in one and two cases respectively. In making the anastomoses non-absorbable sutures were employed in eight and the Murphy button in four cases.

TABLE II

ORIGINAL OPERATION

	Cases
Gastrojejunostomy	
Posterior	45
Anterior	40
Type not stated	7
Total	92
Pyloric resection	
Billroth II	4
Pólya	4
Hofmeister	1
Type not stated	2
Total	11
Combinations with the above	
Entero-anastomosis	16
Pyloric exclusion	5
Roux en Y	2
Excision of ulcer	1
Total	24
Method of making anastomosis	
Non-absorbable suture	8
Murphy button	4

TABLE II. Showing the types of original operation which were followed by gastrojejunal ulcer complicated by acute perforation. Gastrojejunostomy was employed in 92 cases and pyloric resection in 11 cases. Other operative procedures combined with these were used in 24 cases. In making the anastomoses non-absorbable sutures were used in eight cases and the Murphy button in four cases.

Interval between Gastrojejunostomy and Acute Perforation.—The intervals between the performance of gastrojejunostomy and the occurrence of acute perforation of gastrojejunal ulcer were recorded in 101 cases. The shortest interval during which acute perforation occurred was five days and the longest 18 years. In 13 cases (13 per cent) perforation occurred within three months after operation. In 59 cases (58 per cent) it occurred within two years and in 85 cases (84 per cent) within five years. In only 16 cases (16 per cent) did perforation occur as late as from five to 18 years.

Pathology of Acute Perforation of Gastrojejunal Ulcer.—In the material collected for this study there are 120 acute perforations recorded in the 103 cases. Numerically, therefore, the perforations fall into two groups, one group representing multiple and the other single perforations.

Multiple Perforations.—The multiple perforations, likewise, fall into two groups. One subgroup consists of multiple simultaneous perforations and the other of multiple successive perforations (Table III). In three cases multiple (two) perforations were found to exist simultaneously at operation or autopsy. In one case the site of the perforations was not stated but in each of the other two cases the perforations were situated at the anastomosis and in the jejunum.

TABLE III

MULTIPLE ACUTE PERFORATIONS

Multiple (2) Simultaneous Perforations

Author	Cases	Site
Key	1	Not stated
Massie	1	Anastomosis and efferent limb
Charbonnel and Leuret	1	Anastomosis and jejunum
Total	3	

Multiple Successive Perforations

Author	Cases	Site		Total Acute Perforations
		Peptic Ulcer (Gastric or duodenal)	Gastrojejunal Ulcer	
Battle	1	1	3	4
Maylard	1	1	2	3
Henry	1	1	4	5
Steinberg	1	1	2	3
Riess	1	0	2	2
Robinson	1	0	2	2
Nixon and Lowry	1	0	3	3
Woodside	1	0	2	2
Dineen	1	0	2	2
Hinton and Church	1	0	2	2
Schilling	1	0	2	2
Totals	11	4	26	30

TABLE III.—Tabulates the three cases of multiple simultaneous and 11 cases of multiple, successive, acute perforations of gastrojejunal ulcer.

In 11 cases multiple (from two to five) perforations were found to have occurred at different times. In four of these cases acute perforation of peptic (gastric or duodenal) ulcer preceded acute perforation of gastro-

jejunal ulcer. In this group of 11 cases acute perforation of gastrojejunal ulcer occurred twice in eight cases, three times in two cases, and four times in one case. There were four acute perforations of peptic ulcer which, combined with 26 acute perforations of gastrojejunal ulcer, comprise a total of 30 acute perforations of ulcer in the 11 patients. Acute perforation of peptic ulcer followed by single perforation of gastrojejunal ulcer has been discussed under primary pathology.

Single Perforations.—Single perforation of gastrojejunal ulcer occurred in 89 cases. The important pathologic features in these cases are included in the following section.

Site of Perforation.—In the 120-acute perforations of gastrojejunal ulcer in this study the site was recorded in 96 instances (Table IV). The perforation was in the stomach three times, at the anastomosis in 19 instances, and in the jejunum 74 times. Jeunal perforation occurred in the afferent limb ten times, opposite the anastomosis 19 times and in the efferent limb 45 times.

TABLE IV

SITE OF PERFORATION

	Cases	
	Number	Per Cent
Stomach	3	3.1
At anastomosis	19	19.7
Jejunum		
Afferent limb	10	10.4
Opposite anastomosis	19	19.7
Efferent limb	45	46.8
Totals	96	99.7

ASSOCIATED PATHOLOGY

	Cases
Multiple ulcers	17
Hemorrhage	14
Gastrojejunocolic fistula	6
Gastric ulcer	2
Abscess	2
Obstruction	
At anastomosis	2
At pyloris	1
Total	44

TABLE IV.—Showing the site of perforation of and the pathology associated with acute perforation of gastrojejunal ulcer.

TABLE V

TREATMENT

Primary Operation	Deaths			Secondary Operation Required	
Operation	No. of Cases	Cases	Per Cent Mortality	No of Cases	Operation
Not performed	22	2	90.9	2	Drainage of abscesses
Suture					
Simple suture	51	9	17.6	4	Disconnection of gastrojejunostomy; Gastroduodenostomy
With excision of ulcer	6	0	0.0		Pylorectomy Roux; Pylorectomy Pólya
With entero-anastomosis	5	0	0.0	1	Pyloric resection
With jejunostomy	1	1	100.0		
Totals	63	10	15.8		
Disconnection of gastrojejunostomy	4	0	0.0	1	Posterior gastrojejunostomy
Gastrojejunostomy					
Type not stated	4	1	25.0		
Anterior	2	0	0.0	1	Disconnection of gastrojejunostomy
With entero-anastomosis	1	1	100.0		
Totals	7	2	28.5		
Pyloric resection					
Billroth II	4	0*	0.0		
Pólya	3	0	0.0		
Reichl-Roux	3	0	0.0		
Billroth I	1	0	0.0		
Mikulicz-Reichl	1	0	0.0		
Kronlein-Roux	1	0	0.0		
Hoffmeister	1	0	0.0		
Type not stated	3	1	33.3		
Totals	17	1	5.8		
Other operations					
Type not stated	2	?	†		
Appendectomy	1	1	100.00		
Resection of jejunum, anastomosis en Y and pyloric exclusion with ligature	1	0	0.0		
Totals	4	?	?		

* Death or recovery in 1 case not stated

† Death or recovery not stated

TABLE V.—Comparison of the different types of surgical treatment with results in 117 acute perforations of gastrojejunal ulcer. The mortality in the untreated cases reached the high point of 90.9 per cent whereas in simple disconnection of gastrojejunostomy the mortality was 0.0 per cent.

Associated Pathology.—Complicating pathology was associated with acute perforation of gastrojejunal ulcer in 44 cases. Multiple jejunal ulcers were present in 17 cases. In von Eiselsberg's case there were five jejunal ulcers in addition to a gastrojejunocolic fistula. Hemorrhage had complicated gastrojejunal ulcer in 14 cases. In six gastrojejunocolic fistulae were present. Gastric ulcer, intraperitoneal abscess and obstruction of the anastomotic stoma each was present in two cases and pyloric obstruction was present in one case.

Autopsy.—Autopsy was performed in 25 cases.

TREATMENT.—In the 22 cases in which primary operation was not performed there were 20 deaths representing a mortality of 90.9 per cent. In both of the patients who recovered secondary drainage of intraperitoneal abscesses was required. Simple suture of the perforation was carried out in 51 cases with nine deaths and a mortality of 17.6 per cent. Simple suture was combined with other procedures in 12 cases with one death. In 63 cases wherein simple suture of the perforation was carried out with or without other procedures the total mortality was 15.8 per cent. Disconnection of the gastrojejunostomy restoring the normal sequencial relation of the stomach and intestinte was utilized in four cases with no deaths. Gastrojejunostomy alone or combined with other procedures was used in seven cases with two deaths and a mortality of 28.5 per cent. Pyloric resection with various types of gastro-intestinal anastomosis was applied in 17 cases with only one death, making a mortality of 5.8 per cent. In the ten cases in which the interval between perforation and operation was mentioned, it was recorded as from immediately up to six hours in seven cases, from seven to 12 hours in two cases and on the next day in one case. In the case in which death resulted, the operation was performed 11 hours after perforation. In four cases other operations ranging from appendectomy to resection of the jejunum with a Y anastomosis and pyloric exclusion by ligature were used. As recovery or death was not recorded in two of these information concerning the mortality in this group is not complete (Table V).

MORTALITY.—Among the 117 acute perforations of gastrojejunal ulcer in this study the outcome (recovery or death) was not recorded in three instances. In the remaining 114 acute perforations there were 34 deaths, representing a gross mortality of 29.8 per cent.

CASE REPORTS

Case 1.—(Toland and Thompson). Male, aged 42. In 1921, onset of epigastric pain relieved by food and soda. Excision of duodenal ulcer, posterior gastrojejunostomy and cholecystostomy in 1924. Pain recurred six months later. In 1927, medical treatment for duodenal ulcer. In 1928, sudden severe pain in the epigastrium later shifting to the suprapubic region and referred to both shoulders. Physical findings consisted of retracted abdomen with generalized tenderness and board-like rigidity, dulness in the flanks but liver dulness was not obliterated. Diagnosis: Perforated duodenal or jejunal ulcer.

Operation three hours after onset of pain disclosed a large quantity of free fluid

in the pelvis and beneath the great omentum. A perforated ulcer 1.5 cm. in diameter was found on anterior wall of jejunum opposite the anastomosis. The scar of a healed ulcer was situated on the anterior wall of stomach 4 cm. proximal to the pylorus. Pylorus patent. Disconnection of gastrojejunostomy, closure of stomach and resection of a portion of ulcer bearing jejunum 12 cm. long with end-to-end anastomosis by suture. Closure of abdomen with drainage.

Pathologic Report.—Specimen of intestine 6 cm. long. Circular opening 14 Mm. in diameter with smooth mucosal edges apparently a gastrojejunostomy opening. Three cm. directly anterior to this opening is a second opening measuring 9 × 12 Mm. extending through the entire wall of the intestine with indurated and undermined mucosal edges typical of a peptic ulcer. Serosa moderately thickened about the opening and covered by a delicate layer of fibrin. No evidence of malignancy. Gross Diagnosis: Perforated jejunal ulcer. Recovery.

Three months after operation patient was readmitted to hospital for observation for recurrent epigastric pain. Fluoroscopic examination disclosed a dilated stomach and tendency for the barium to collect in the dilated duodenal loop. In 1929, patient was readmitted for persistent pain, after having vomited one quart of coffee ground material, followed by two copious tarry defecations. Red blood corpuscles, 1,480,000. Night retention of from 250 to 650 cc. Medical treatment for two months including transfusion of blood.

Second Operation.—Eighteen months after acute perforation of gastrojejunal ulcer: Many adhesions. Firm mass at pylorus. Head of pancreas hard and enlarged. Posterior no-loop gastrojejunostomy with sutures. General condition of patient too poor to risk resection of tumor at pylorus. Recovered. Readmitted to hospital two months postoperative because of persistence of pain in lower abdomen. Improved under medical treatment. When last examined in October, 1930, patient still complained of pain in the epigastrium. Medical treatment was advised.

Case 2.—(Toland and Thompson). Male, aged 36. In 1917, onset of periodic gnawing pain in the epigastrium appearing two to three hours after meals and relieved by vomiting. Diagnosis: Peptic ulcer. Medical treatment with relief for six months. In 1928, while eating, experienced severe abdominal pain.

Operation six hours after onset of pain disclosed a perforated duodenal ulcer. Sutured. In 1929, posterior gastrojejunostomy with partial relief of symptoms. In 1934, sudden attack of dizziness and vomiting of bright red blood, melena. Medical treatment. Three months later, one and one-half hours after a meal of soft food, patient experienced a sudden severe epigastric pain. Generalized abdominal rigidity was found on physical examination. Diagnosis: Perforated peptic ulcer.

Second operation three and one-half hours after onset of pain disclosed many adhesions but no evidence of peritonitis anterior to the great omentum. Entire region beneath the transverse colon filled with milky fluid. Multiple lesions in the omentum resembling fat necrosis. Perforation of anterior wall of jejunum directly below anastomosis 2 Mm. in diameter. Suture. Closure with drainage. Recovery. On follow-up five months postoperative, patient found to experience ulcer pain at intervals. Diagnosis: Marginal or jejunal ulcer.

Case 3.—(Toland and Thompson). Male, aged 65. In 1924, onset of pain following meals, relieved by soda. In 1929, vomiting of practically all food except fluids. Diagnosis: Peptic ulcer with obstruction. Posterior no-loop gastrojejunostomy for duodenal ulcer with obstruction. In 1934 after severe agonizing epigastric pain readmitted to the hospital where a diagnosis of perforated peptic ulcer was made. The patient stated that he had been free from symptoms until three weeks prior to this admission.

Operation disclosed perforated ulcer of afferent limb of jejunum. Mass in first portion of duodenum had disappeared. Suture and entero-anastomosis. Recovery.

Case 4.—(Toland and Thompson). Male, aged 34. In 1929, gnawing epigastric

pain after meals relieved by food, of three weeks' duration when patient experienced a sudden sharp stabbing epigastric pain. Examination disclosed moist cold skin and rigid abdomen. Diagnosis: Perforated peptic ulcer.

Operation disclosed ulcer anterior wall of pylorus with perforation 1 cm. in diameter. Suture and posterior gastrojejunostomy. Relief until 1934 when there was a recurrence of severe abdominal pain. Abdomen board-like and tender throughout. Diagnosis: Perforated peptic ulcer. Operation: Perforated ulcer at the margin of the gastrojejunostomy opening. Clear fluid in peritoneal cavity. Closure of perforation with sutures. Drainage of peritoneal cavity. On sixth day postoperative evisceration through a small opening in the incision. Secondary closure. Patient died on sixteenth day postoperative Autopsy: Generalized peritonitis. Ulcer in greater curvature of stomach communicating with jejunum. Bilateral fibrinous pleuritis.

Case 5.—(Toland and Thompson). Male, aged 37. Gastrojejunostomy eight years ago for gastric ulcer. Recent examination for recurrence of symptoms at the Mayo Clinic resulted in a diagnosis of marginal ulcer and reactivation of duodenal ulcer. In September, 1935, patient experienced a sudden pain in the right upper quadrant of the abdomen. Examination disclosed a flat, rigid, tender anterior abdominal wall and obliteration of liver dulness. Diagnosis: Acute perforation of duodenal ulcer.

Operation disclosed an active duodenal ulcer, which was not ruptured, and an acute perforation of an ulcer in the efferent limb of the jejunum. Gentle manipulation of the jejunum caused a separation throughout half of the circumference of the anastomosis. Disconnection of gastrojejunostomy with suture of gastric and jejunal openings and restoration of normal anatomy. Recovery.

Case 6.—(Hinton). Male, aged 45. Posterior gastrojejunostomy for duodenal ulcer in 1920. In 1933, acute perforation of ulcer of efferent limb of jejunum. Simple suture. Recovery. Recurrence of pain. Further surgery advised.

Case 7.—(Hinton). Male, aged 55. Posterior gastrojejunostomy in 1929. On February 25, 1936, acute perforation of gastrojejunal ulcer situated in the efferent limb. Simple suture. Recovery.

Case 8.—(Storey). Male, aged 44. In 1924, posterior gastrojejunostomy for duodenal ulcer with obstruction. Relief for 11 months, when pain in the epigastrium associated with melena appeared. Gastric analysis disclosed hyperacidity and fluoroscopic examination with a barium meal revealed an accessory pocket near the stoma suggestive of ulcer. In 1925, operation disclosed a cavity filled with brown fluid situated between the jejunum and transverse mesocolon into which a perforation of the jejunum emptied. A piece of silk three inches long was removed from the neighborhood of the ulcer. The duodenum was contracted to the diameter of a little finger. Gastrojejunostomy disconnected, openings closed, anterior gastrojejunostomy performed. Recovery.

Case 9.—(Storey). Male, aged 52. In 1923, after having had epigastric pain for 12 months, anterior gastro-enterostomy and entero-anastomosis were performed. In 1928, sudden severe pain in the region of the umbilicus.

Operation disclosed adhesions between the omentum, stomach and abdominal wall and free fluid in the peritoneal cavity. A small perforation in the efferent limb of the jejunum was sutured. Recovery.

Case 10.—(Storey). Male, aged 47. In 1929, posterior gastrojejunostomy for duodenal ulcer with obstruction. In 1932, perforated bleeding jejunal ulcer opposite the stoma closed with sutures. Recovery.

Discussion.—In addition to the contribution of ten new cases of acute perforation of gastrojejunal ulcer to the literature, this study of 103 cases has emphasized a number of significant facts. They will be discussed under three broad headings; namely, etiology, pathology, and treatment.

Etiology.—This study reemphasizes that while acute perforation of gastro-

jejunal ulcer may occur at any period from infancy to old age, the fact that 85 per cent of cases were found to occur from the third through the fifth decade indicates that the condition is distinctly a disease of early and middle adult life. By a similarly large percentage of cases the condition also is seen to predominate in males.

An analysis of the data on the primary pathology has brought out two important facts. First, it confirms the prevailing impression that acute perforation of gastrojejunal ulcer occurs most frequently following peptic ulcer. What is more important, however, is that it illustrates how perforation of gastrojejunal ulcer may occur not only after malignancy of the stomach but also as an untoward sequela to gastrojejunostomy when performed in such benign conditions as gastric erosions, gastroptosis or even in the absence of pathology. These points serve to emphasize further the inadvisability of application of this operation in functional conditions or when pathology is not demonstrable or is indefinite.

The second important fact is that pyloric stenosis was recorded as being the primary pathology in 18 cases. It will be noted that most of these cases were reported by early writers. One of us (H.L.T.[41]), in collaboration with Judd, has shown that while primary pyloric stenosis exists, its occurrence among adults is rare. While it is probable, therefore, that many if not all of the cases in this group were in reality pyloric or duodenal ulcer with secondary stenosis, the absence from the case reports of data which would enable us to distinguish the two forms made it necessary for us to recognize two groups. Had we combined the groups there would have been 38 cases or as high as 36.8 per cent of the entire series wherein acute perforation of gastrojejunal ulcer followed pyloric obstruction. This is of special interest in view of Wilkie's[96] recent statement that gastrojejunostomy should be performed only in cases of pyloric obstruction.

Analysis of the data on complications of the primary pathology is also significant. It is seen that complications were present in 63 per cent of the cases in which gastrojejunostomy was used. Acute perforation of peptic ulcer outnumbers peptic ulcer with obstruction by 5 per cent and becomes therefore the most common complication to be followed by acute perforation of gastrojejunal ulcer. By carrying the analysis to the point of distinguishing between the tendencies manifest by peptic ulcer we find that the liability to reactivation, as indicated by acute and chronic perforation and by erosion of blood vessels, outstrips the tendency to inactivity as shown by scarring and obstruction in a case ratio of 37 to 20.

The secondary nature and occurrence of gastrojejunal ulcer brings our consideration of the types of original operation under the heading of etiology. As gastrojejunostomy *per se* is an essential prerequisite to the formation of gastrojejunal ulcer, it is necessary for us to consider only the various types of this operation and the effects of the operations which were used in combination with it.

The comparative frequency of acute perforation of gastrojejunal ulcer

with respect to anterior or posterior gastrojejunostomy is striking. During the early history of gastrojejunal ulcer when anterior gastrojejunostomy was used more often than at present, the impression prevailed among students of this subject that perforating gastrojejunal ulcer was more commonly a sequela to anterior rather than to posterior gastrojejunostomy. Our figures reveal, however, that the condition is even more common after posterior gastrojejunostomy, so that the recorded cases now outnumber the others by 45 to 40.

The occurrence of acute perforation of gastrojejunal ulcer after pyloric resection is of interest from a physiologic standpoint. The impression is prevalent among physicians that pylorectomy reduces the secretion of hydrochloric acid. It has been shown experimentally by one of us (H.L.T[90]) that this depends also on the amount of stomach mucosa resected and on the type and quantity of food consumed. Also of interest from a physiologic standpoint is the large number of acute perforations of gastrojejunal ulcer which occurred after entero-anastomosis. It would appear from these figures that any procedure which shunts the alkaline duodenal juices around the gastrojejunal stoma predisposes strongly to the formation of gastrojejunal ulcer. The use of mechanical devices such as the Murphy button and of non-absorbable sutures appear also to be predisposing factors.

Our findings that 58 per cent of acute perforations of gastrojejunal ulcer occur within two years are in agreement with the recent statements of Judd and Hoerner[40] in this regard.

Pathology.—The pathology of acute perforation of gastrojejunal ulcer especially with respect to multiple perforations is of interest. In view of the fact that multiple gastrojejunal ulcers are relatively common, it is not unusual that a few cases of multiple simultaneous perforations should be encountered.

From the clinical and therapeutic standpoint, one of the most important phases of this study is that of multiple successive acute perforations. The striking tendency of peptic and gastrojejunal ulcers to perforate is amply illustrated by acute perforation of peptic ulcer having preceded acute perforation of gastrojejunal ulcer in 24 cases and by the latter having occurred from two to four times in 11 cases. From this apparent total of 35 cases, four cases must be subtracted as common to both groups, leaving a real total of 31 cases which is 30 per cent of the entire series. When we note that gastrojejunocolic fistula was also associated with acute perforation in six cases, the point is even more striking. This evidence appears to be sufficiently convincing to justify the dictum that gastrojejunostomy should never be performed in unobstructed acute perforation of peptic ulcer. The tendency of peptic and gastrojejunal ulcers to perforation is so strong that in a significant proportion of cases it manifests itself over a period of weeks or years in multiple perforations.

The site of perforation having been found to be more frequent about the artificial stoma, and opposite or distal to it in the jejunum, supports the tissue susceptibility theory of Ochsner[63] and indicates that the corrosive

TABLE VI

SUMMARY OF REPORTED CASES OF GASTROJEJUNAL ULCER*

No.	Reported by Author	Year	Age in Years	Sex	Original Pathology	Original Operation	Interval	Pathology of Gastrojejunal Ulcer	Operation for Gastrojejunal Ulcer	Result	Remarks
1	Braun[6]	1899	25	M.	Gastric ulcer, obstruction	Post. gastrojejunostomy	11 mos.	Jejunum, efferent	None	Death	
2	Hahn [39]	1899	—	—	Pyloric stenosis	Ant. gastrojejunostomy	1 yr.	Jejunum, afferent	None	Death	
3	Korte[46]	1900	30	M.	Gastric ulcer, obstruction	Ant. gastrojejunostomy	3 yrs.	Jejunum, efferent	Appendectomy	Death	
4	Steinthal[87]	1900	44	M.	Chronic gastritis, hemorrhage	Post. gastrojejunostomy, Murphy button	10 da.	Jejunum, efferent	None	Death	
5	Goepel[25]	1902	—	M.	Pyloric stenosis	Ant. gastrojejunostomy	13 mos.	—	None	Death	See also Tiegel
6	Goepel[25]	1902	—	M.	Pyloric stenosis	Ant. gastrojejunostomy	4 mos.	—	Suture	Recovery	First case operated upon
7	Mikulicz[59]	1903	5 mos.	F.	Congenital hypertrophic stenosis, obstruction	Ant. gastrojejunostomy, entero-anastomosis	82 da.	Jejunum, afferent. Multiple ulcers. Hemorrhage	None	Death	
8	Brentano[7]	1903	26	F.	Pyloric stenosis	Ant. gastrojejunostomy, entero-anastomosis	1 yr.	Anastomosis	New gastro-jejunostomy	Recovery	
9	Cackovic[11]	1903	30	M.	Pyloric stenosis, obstruction	Post. gastrojejunostomy	5 da.	Jejunum, efferent Multiple ulcers	None	Death	
10	Tiegel[92]	1904	34	M.	——	Ant. gastrojejunostomy	9 mos.	—	Suture	Recovery	
11	Dudgeon and Sargent[17]	1905	30	M.	Gastric ulcer	——	22 mos.	—	Suture	Recovery	
12	Battle[2]	1906	30	M.	Gastric ulcer, obstruction, hemorrhage	Ant. gastrojejunostomy, Murphy button	22 mos.	—	Suture	Recovery	
13	I† Battle[2]	1906	37	F.	Gastric ulcer, obstruction, acute perforation	Ant. gastrojejunostomy	13 mos.	Jejunum, efferent	Suture	Recovery	
	II Battle[2]	1906		F.	Gastric ulcer, obstruction, acute perforation	Ant. gastrojejunostomy	23 mos.	Anastomosis	Suture	Recovery	
	III Romanis[75]	1918		F.	Gastric ulcer, obstruction, acute perforation	Ant. gastrojejunostomy	13 yrs.	Jejunum, efferent	Suture	Recovery	
14	Delaloye[14]	1906	41	M.	Duodenal ulcer, obstruction	Ant. gastrojejunostomy	6 yrs.	Anastomosis	None	Death	
15	Graser[27]	1906	—	F.	Gastric ulcer, obstruction	Post. gastrojejunostomy, Murphy button	4 yrs.	Jejunum, efferent, hemorrhage	None	Death	
16	Eiselsberg[20]	1906	—	M.	Gastric ulcer	Billroth II	1 yr.	Stomach	None	Death	
17	Paterson[64, 65, 66]	1906	48	M.	Pyloric stenosis	Post. gastrojejunostomy	2 yrs.	Jejunum, efferent	None	Death	
18	Paterson[64, 65, 66]	1906	51	M.	Pyloric stenosis	Ant. gastrojejunostomy	2½ yrs.	Jejunum, opposite anastomosis	None	Death	
19	Key[48]	1907	45	F.	Gastric ulcer, obstruction, hemorrhage, perforation into pancreas	Ant. gastrojejunostomy Y	7 yrs.	Anastomosis	Excision of ulcer, enlargement of anastomosis, suture	Recovery	
20	Key[45]	1907	25	F.	Carcinoma stomach	Billroth II	10 da.	Multiple ulcers, 2 simultaneous perforations	None	Death	

* In the compilation of the data in this table of collected cases the original case report was consulted in every instance.

† The Roman numeral indicates the number of each perforation as it occurred in cases in which multiple successive perforations took place.

21	Edington[18]	1907	46	M.	Duodenal ulcer, obstruction	Ant. gastrojejunostomy	7 yrs.	Jejunum, efferent	Suture	Death	
22	Hamann[31]	1907	48	M.	Gastric erosions	Ant. gastrojejunostomy, Murphy button, Entero-anastomosis	26 da.	Jejunum, efferent	None	Death	
23	Jainu[38]	1907	—	M.	—— Hemorrhage	Post. gastrojejunostomy	11 mos.	Jejunum, opposite anastomosis	None	Death	
24	Jensen[39]	1908	54	M.	Pyloric stenosis	Post. gastrojejunostomy	13 mos.	Jejunum, efferent	None	Death	
25	Lion and Moreau[55]	1909	43	M.	Pyloric stenosis	Post. gastrojejunostomy	8 yrs.	Jejunum, afferent, hemorrhage, jejunocolic fistula	None	Death	
26	Eiselsberg[21]	1910	40	M.	Gastric ulcer	Post. gastrojejunostomy	2 mos.	Jejunum, opposite anastomosis, multiple (5) ulcers, jejunocolic fistula	New gastro-jejunostomy, entero-anastomosis	Death	
27	I Maylard[57]	1910	51	F.	Gastric ulcer, acute perforation	Ant. gastrojejunostomy	8 mos.	Jejunum, efferent	Suture	Recovery	
	II Maylard[57]	1910	51	F.	Gastric ulcer, acute perforation	Ant. gastrojejunostomy	3 yrs.	Jejunum, efferent	Suture	Recovery	
28	Battle[3]	1910	39	M.	Gastric ulcer, obstruction, acute perforation	Ant. gastrojejunostomy	2 yrs.	Anastomosis	Suture	Recovery	
29	Petren[67]	1911	37	M.	Gastric ulcer, acute perforation	Post. gastrojejunostomy, entero-anastomosis	9 mos.	Anastomosis	Excision of ulcer, suture	Recovery	
30	Petren[68]	1911	18	M.	Gastric ulcer, obstruction, hemorrhage	Post. gastrojejunostomy	5 da.	Jejunum, afferent, multiple ulcers	None	Death	
31	Hitzrot[36]	1911	26	M.	Gastric ulcer, obstruction	Ant. gastrojejunostomy, Roux Y	7 yrs.	Jejunum, efferent	Suture	Recovery	
32	Moynihan[61]	1912	29	M.	Pyloric stenosis	Post. gastrojejunostomy	7 da.	Jejunum, opposite anastomosis	Suture	Death	
33	Rowlands[76, 77]	1912	30	M.	Pyloric stenosis	Ant. gastrojejunostomy	5 yrs.	Jejunum, efferent	Suture	Recovery	Secondary operation: Disconnection
34	Lindner[54]	1912	50	F.	——	——	—	——	None	Death	Secondary operation: Gastrojejunostomy Died 2 days post-operative of acute perforation at first gastrojejunostomy
35	Lindner[54]	1912	—	M.	——	——	6 yrs.	Anastomosis	Suture	Recovery	
36	Hartmann[32]	1913	41	M.	Pyloric stenosis	Post. gastrojejunostomy	6 yrs.	Anastomosis	Suture	Recovery	
37	Schmilinsky[79]	1918	—	—	Duodenal ulcer	Post. gastrojejunostomy, pyloric exclusion	22 mos.	——	Suture	Recovery	
38	Schmilinsky[79]	1918	—	—	Duodenal ulcer	Post. gastrojejunostomy, pyloric exclusion	16 mos.	Jejunum, efferent	Suture	Death	
39	Haberer[28, 29]	1918	40	M.	Duodenal ulcer	Post. gastrojejunostomy, pyloric exclusion	58 da.	—— Multiple ulcers, (4) colic fistula	Suture	Death	
40	Wright[101]	1919	39	M.	Duodenal ulcer, obstruction	Ant. gastrojejunostomy	19 mos.	Jejunum, efferent	Suture	Recovery	

COLLECTED CASES—*Continued*

No.	Reported by Author	Year	Age in Years	Sex	Original Pathology	Original Operation	Interval	Pathology of Gastrojejunal Ulcer	Operation for Gastrojejunal Ulcer	Result	Remarks
41	Roberts[73]	1920	34	M.	Pyloric stenosis, acute perforation	Post. gastrojejunostomy	2½ mos.	Jejunum, afferent	Suture	Recovery	
42	Polya[69]	1920	36	M.	Duodenal ulcer	Post. gastrojejunostomy, pyloric exclusion	10 mos.	Jejunum, opposite anastomosis, multiple (4) ulcers	Suture	Death	
43	I Henry[33]	1921	36	M.	Gastric ulcer, acute perforation	Ant. gastrojejunostomy	5 mos.	Jejunum, opposite anastomosis	Suture	Recovery	
	II Henry[33]	1921	—	M.	Gastric ulcer, acute perforation	Ant. gastrojejunostomy	2½ yrs.	Jejunum, efferent	Suture	Recovery	
	III Henry[33]	1921	—	M.	Gastric ulcer, acute perforation	Ant. gastrojejunostomy	4½ yrs.	——	Suture	Recovery	
	IV Henry[33]	1921	—	M.	Gastric ulcer, acute perforation	Ant. gastrojejunostomy	6 yrs.	Anastomosis	Excision of ulcer, suture	Recovery	
44	Urrutia[94]	1921	35	M.	Duodenal ulcer, acute	Post. gastrojejunostomy	14 mos.	Jejunum, efferent	Suture	Recovery	Secondary operation: Roux resection
45	Haberer[29]	1922	34	—	Gastric ulcer	Post. gastrojejunostomy	4 yrs.	——	Suture, jejunostomy	Death	
46	Brutt[9]	1922	18	M.	Pyloric stenosis	Ant. gastrojejunostomy	3 yrs.	——	New gastro-jejunostomy	Recovery	
47	Brutt[9]	1922	40	M.	Pyloric stenosis	Ant. gastrojejunostomy	6 yrs.	Jejunum, efferent, hemorrhage	Suture	Death	
48	McKelvey[58]	1922	44	M.	Gastric ulcer	Ant. gastrojejunostomy	18 yrs.	Anastomosis	Disconnection	Recovery	
49	Massie[56]	1924	28	M.	Duodenal ulcer	Ant. gastrojejunostomy	16 mos.	Multiple simultaneous perforations (2): Anastomosis, jejunum, afferent	Suture	Recovery	Secondary operation: Disconnected
50	Bundschuh[8]	1924	49	M.	Gastric ulcer	Ant. gastrojejunostomy, entero-anastomosis	14 yrs.	Jejunum, efferent, multiple ulcers (2): Acute perforation, Jejunocolic fistula	Pyloric resection, Mikulicz-Reichel anastomosis	Recovery	
51	I Steinberg[85]	1924	36	M.	Duodenal ulcer, acute perforation	Post. gastrojejunostomy	—	Jejunum, efferent, multiple ulcers	Suture	Recovery	
	II Steinberg[86]	1936	—	M.	——	——	—	——	Suture Jejunum	Recovery	
52	Schwarz[80]	1925	—	M.	Gastric ulcer	Ant. gastrojejunostomy	3 yrs.	Jejunum, opposite anastomosis	None	Death	
53	Schwarz[80]	1925	39	M.	Gastric ulcer	Ant. gastrojejunostomy	1 yr.	Anastomosis, multiple ulcers	Pyloric resection, Reichel-Roux anastomosis	Recovery	
54	Schwarz[80]	1925	33	F.	Duodenal ulcer	Ant. gastrojejunostomy	1½ yrs.	Jejunum, efferent	Pyloric resection, Reichel-Roux anastomosis	Recovery	
55	I Riess[72]	1925	27	M.	Gastric ulcer, perforation into pancreas	Ant. gastrojejunostomy	86 da.	Jejunum, opposite anastomosis	Excision of ulcer, suture	Recovery	

	II Reiss[72]	1925	—	M.	——	——	9 mos.	Jejunum, afferent, multiple ulcers	Pyloric resection, Billroth II. anastomosis	Recovery	
56	Charbonnel and Leuret[12]	1925	60	M.	Gastric ulcer, obstruction	Post. gastrojejunostomy	3 yrs.	Multiple (2) simultaneous perforations: Anastomosis, jejunum, efferent	Suture	Death	
57	Zeno[163]	1926	23	M.	Duodenal ulcer, acute perforation	Post. gastrojejunostomy	5 mos.	Anastomosis, multiple ulcers	Ant. gastrojejunostomy	Recovery	Secondary operation: Disconnection both gastrojejunostomies
58	Ashcroft[1]	1926	38	M.	Duodenal ulcer, acute perforation	Post. gastrojejunostomy	19 mos.	Jejunum, efferent	Suture, entero-anastomosis	Recovery	Secondary operation: Pyloric resection
59	Ashcroft[1]	1926	37	M.	Duodenal ulcer	Post. gastrojejunostomy	9 mos.	Jejunum, efferent	Excision of ulcer, suture	Recovery	
60	Winkelbauer[98]	1926	22	M.	Duodenal ulcer, hemorrhage, acute perforation	Ant. gastrojejunostomy	10 mos.	Jejunum, opposite anastomosis	Pyloric resection, Pólya anastomosis	Recovery	
61	Kunz[47]	1926	34	M.	Pyloric stenosis	Ant. gastrojejunostomy, entero-anastomosis	16 mos.	——	Pyloric resection	Recovery	
62	Kunz[47]	1926	31	M.	No ulcer found	Ant. gastrojejunostomy	16 mos.	Jejunum, opposite anastomosis	Pyloric resection, Reichel-Roux anastomosis	Recovery	
63	Delor y Castro[15]	1926	49	M.	Duodenal ulcer	—— Gastrojejunostomy	2 yrs.	Anastomosis	Pyloric resection	Death	
64	Spath[88]	1927	33	M.	Duodenal ulcer	Ant. gastrojejunostomy, entero-anastomosis	7 mos.	Jejunum, opposite anastomosis	Pyloric resection, Billroth I	Recovery	
65	Jura[42]	1927	37	M.	Duodenal ulcer, hemorrhage	Post. gastrojejunostomy	6 da.	Jejunum, efferent	None	Death	
66	Brutt[10]	1927	40	M.	—— Acute perforation	Billroth II	1 yr.	—— Multiple ulcers.	Suture	Death	
67	Leveuf[49]	1927	47	M.	——	Post. gastrojejunostomy	5 yrs.	Jejunum, efferent	Resection of jejunum, anastomosis Y, pyloric exclusion with ligature	Recovery	
68	Porzelt[70]	1928	31	M.	Duodenal ulcer, acute perforation	Ant. gastrojejunostomy, entero-anastomosis	7½ mos.	Jejunum, opposite anastomosis	Pyloric resection, Kronlein-Roux anastomosis	Recovery	
69	I Robinson[74]	1928	26	M.	Duodenal ulcer	Billroth II	7 mos.	——	Suture	Recovery	
	II Robinson[74]	1928	—	M.	——	——	1 yr.	Jejunum, efferent	Suture	Recovery	
70	Ehrlich[19]	1928	24	M.	Gastric ulcer, doudenal ulcer, acute perforations (2) simultaneous	Post. gastrojejunostomy	4½ mos.	Jejunum, efferent	Suture	Recovery	
71	I Nixon and Lowry[62]	1928	36	M.	Gastric ulcer, hemorrhage	Post. gastrojejunostomy	15 mos.	Stomach	Suture	Recovery	
	II Nixon and Lowry[62]	1928	—	M.	——	——	5 yrs.	Jejunum, efferent	Suture	Recovery	
	III Nixon and Lowry[62]	1928	—	M.	——	——	6 yrs.	Stomach, multiple ulcers	Suture	Recovery	Secondary operation: Pólya resection

COLLECTED CASES—*Continued*

No.	Reported by Author	Year	Age in Years	Sex	Original Pathology	Original Operation	Interval	Pathology of Gastrojejunal Ulcer	Operation for Gastrojejunal Ulcer	Result	Remarks
72	Just[43]	1929	29	M.	Duodenal ulcer, acute perforation	Ant. gastrojejunostomy, entero-anastomosis	10 mos.	Jejunum, opposite anastomosis	Pyloric resection, Pólya anastomosis	Recovery	
73	Fromme[24]	1929	41	M.	Duodenal ulcer, acute perforation	Ant. gastrojejunostomy, entero-anastomosis	9 mos.	Jejunum, opposite anastomosis	Pyloric resection, Billroth II anastomosis	Recovery	
74	Fromme[24]	1929	32	M.	Duodenal ulcer, acute perforation	Ant. gastrojejunostomy, entero-anastomosis	77 da.	——	Pyloric resection, Billroth II anastomosis	—	
75	I Woodside[100]	1931	16	M.	Duodenal ulcer	Post. gastrojejunostomy	4 yrs.	Jejunum, efferent	Suture	Recovery	
	II Woodside[100]	1931	—	M.	——	——	6 yrs.	Jejunum, afferent	Suture	Recovery	
76	Steden[84]	1931	24	M.	Gastroptosis	Ant. gastrojejunostomy, entero-anastomosis	7 mos.	Jejunum, efferent	Pyloric resection	Recovery	
77	Steden[84]	1931	21	M.	Pyloric stenosis, acute perforation	Ant. gastrojejunostomy, entero-anastomosis	8 yrs.	Jejunum, afferent pyloric stenosis	Pyloric resection, Billroth II anastomosis	Recovery	
78	Steden[84]	1931	30	M.	Gastric ulcer, acute perforation	Pyloric resection	4 yrs.	Jejunum, opposite anastomosis, multiple ulcers, jejunocolic fistula	Pyloric resection, Pólya anastomosis	Recovery	
79	Davenport[13]	1931	35	M.	Duodenal ulcer, hemorrhage	Post. gastrojejunostomy	5 yrs.	——	Suture	Recovery	
80	Brandtner and Tonnis[5]	1932	—	M.	Duodenal ulcer, acute perforations (2) consecutive	Pyloric resection	4 mos.	Anastomosis	Suture	Recovery	
81	Bertrand and Etienne-Martin[4]	1933	61	M.	Gastric ulcer, acute perforation	Post. gastrojejunostomy	3½ yrs.	Jejunum, opposite anastomosis	Suture, entero-anastomosis	Recovery	
82	I Dineen[16]	1934	27	M.	Duodenal ulcer, hemorrhage	Pólya resection	4 yrs.	Jejunum, efferent, hemorrhage	Suture	Recovery	
	II Dineen[16]	1934	—	M.	——	——	4½ yrs.	Jejunum, efferent	Suture, entero-anastomosis	Recovery	
83	Singer and Meyer[81]	1934	42	F.	Pyloric stenosis	——	1 yr.	Jejunum, efferent	Suture	Recovery	
84	Singer and Meyer[81]	1934	38	M.	——	Pólya resection	1 yr.	Jejunum, hemorrhage	None	Recovery	Secondary operation: Drainage abscess
85	Singer and Meyer[81]	1934	50	M.	—— Acute perforation	——	3 yrs.	Jejunum, efferent, hemorrhage, gastrocolic fistula	New gastrojejunostomy	Death	
86	Singer and Meyer[81]	1934	49	M.	——	Post. gastrojejunostomy	3 yrs.	Jejunum, stoma obstruction	None	Recovery	Secondary operation: Drainage abscess
87	Singer and Meyer[81]	1934	43	M.	—— Obstruction	——	14 yrs.	Anastomosis	Suture	Recovery	

88	I Hinton and Church[35]	1934	27	M.	Duodenal ulcer, hemorrhage	Pólya resection	4 yrs.	Jejunum, efferent, hemorrhage	Suture	Recovery
	II Hinton and Church[35]	1934	—	M.	——	——	5 yrs.	Jejunum, efferent	Suture, entero-anastomosis	Recovery
89	Lewisohn[51]	1934	53	M.	Duodenal ulcer	Hofmeister resection	3 yrs.	Anastomosis, stoma obstruction	Pyloric resection Hofmeister anastomosis	Recovery
90	I Schilling[78]	1935	31	M.	Duodenal ulcer	Pólya resection, entero-anastomosis	2 yrs.	——	Excision of ulcer, suture, new gastrojejunostomy	Recovery
	II Schilling[78]	1935	—	M.	——	——	2 yrs.	Multiple ulcers	Suture, resection	Recovery
91	Trevor[93]	1935	33	M.	Duodenal ulcer	Post. gastrojejunostomy	18 mos.	——	—	—
92	Trevor[93]	1935	51	M.	——	Post. gastrojejunostomy	8 yrs.	Jejunum, efferent	—	—
93	Wilkinson[97]	1935	32	M.	Gastric ulcer	Post. gastrojejunostomy	14 mos.	Jejunum, hemorrhage		
94	Toland and Thompson	1936	42	M.	Duodenal ulcer	Excision of ulcer, post. gastrojejunostomy	4 yrs.	Jejunum, efferent, hemorrhage	Disconnection	Recovery
95	Toland and Thompson	1936	36	M.	Duodenal ulcer, acute perforation	Post. gastrojejunostomy	5 yrs.	Jejunum, opposite anastomosis	Disconnection	Recovery Secondary operation: Post. gastrojejunostomy
96	Toland and Thompson	1936	65	M.	Duodenal ulcer, obstruction	Post. gastrojejunostomy	3 yrs.	Jejunum, opposite anastomosis, hemorrhage	Suture	Recovery
97	Toland and Thompson	1936	34	M.	Gastric ulcer, acute perforation	Post. gastrojejunostomy	5 yrs.	Jejunum, afferent	Suture, entero-anastomosis	Recovery
98	Toland and Thompson	1936	37	M.	Duodenal ulcer	Post. gastrojejunostomy	8 yrs.	Anastomosis	Suture	Death
99	Hinton	1936	45	M.	Duodenal ulcer	Post. gastrojejunostomy	13 yrs.	Jejunum, efferent	Disconnection	Recovery
100	Hinton	1936	55	M.	——	Post. gastrojejunostomy	7 yrs.	Jejunum, efferent	Suture	Recovery
101	Storey	1936	44	M.	Duodenal ulcer, obstruction	Post. gastrojejunostomy	1 yr.	Jejunum, hemorrhage	Suture	Recovery
102	Storey	1936	52	M.	——	Ant. gastrojejunostomy, entero-anastomosis	5 yrs.	Jejunum, efferent	New ant. gastro-jejunostomy	Recovery
103	Storey	1936	47	M.	Duodenal ulcer, obstruction	Post. gastrojejunostomy	2½ yrs.	Jejunum, opposite anastomosis, hemorrhage	Suture	Recovery

effect of the acid gastric juice plays a highly important rôle in the production and perforation of gastrojejunal ulcer.

Treatment.—The data which we have collected amply illustrate the efficacy of surgical treatment and the futility of expectant treatment. As against a mortality of 90.9 per cent in the untreated cases, the highest mortality in the treated cases was 28.5 per cent. While the numbers of cases in some of the operation groups are admittedly small, the mortality rates, we believe, are significant. Disconnection of gastrojejunostomy appears to be the safest procedure and probably should be carried out when the patency of the pylorus will permit. Simple suture carrying a mortality of 17.6 per cent and requiring more secondary operations than the other procedures is by virtue of its simplicity applicable to the largest number of cases. This fact alone would tend to make the mortality for this operation appear high. Gastrojejunostomy, which has a mortality in this series of 25 to 28.5 per cent, appears, in view of our other findings, as not only ineffectual but perhaps meddlesome, except in cases wherein pyloric obstruction coexists. The most remarkable finding with respect to treatment of acute perforation of gastrojejunal ulcer was the fact that in 17 cases wherein pyloric resection was carried out there was only one death, representing a mortality of 5.6 per cent.

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LATE RESULTS IN ACUTE PERFORATED PEPTIC ULCER TREATED BY SIMPLE CLOSURE

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THIS investigation was undertaken with the primary object of ascertaining late results in cases of acute perforated peptic ulcer treated by simple suture alone. It is based upon a series of 74 such cases operated upon at the Beekman Street Hospital during the years 1926–1932, inclusive. Cases treated by other methods and a few who refused operation are omitted. Otherwise this represents a consecutive series. A postoperative interval of at least 12 months was required in each case considered; in the majority of cases at least three years had elapsed since operation.

In endeavoring to obtain a comprehensive and first hand estimate of late results, an attempt was made by the author to interview and examine each case personally. Of 66 letters sent out for this purpose, 26 patients came in for interview and examination. There were in addition six patients who had recently been seen in our regular follow-up clinic so that we had, through examination, recent information about 32 of the 66 who survived operation, making a total of 48.4 per cent followed over a period of one to six years and recently examined.

To 34 cases with whom a special interview could not be arranged, a questionnaire was sent. Of this number two subsequently came in for examination, and of the remaining 32, 13 replies were received. While it is appreciated that information obtained by questionnaire usually has not the value of that resulting from direct follow-up examination, being at times even misleading, in composing the questionnaire an attempt was made to elicit replies which would be comprehensive and pertinent. It was gratifying to find that the replies were in the main complete, earnestly filled in and in many instances accompanied by letters which gave additional information that proved of value. The author believes that the benefit of this type of late study, seriously conceived, tends to be underestimated.

Adding to the 32 cases recently examined the 13 replies to questionnaire,

TABLE I

FOLLOW-UP STATISTICS

	Number	Per Cent
Total cases surviving operation	66	
Special follow-up examination	26	39.3
Recent examination	6	
Total examined recently	32	48.4
Questionnaire replies	13	
Total late follow-up	45	68.1

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late information was obtained in 45 of the total series of 66 survivors, 68.1 per cent (Table I).

While primarily undertaken to determine late results, analysis of case records in this study revealed many other interesting points which merit some consideration.

Sex.—It was somewhat surprising that all of the 74 patients in this series were males, and it might be added that, in going over all the records of the Beekman Street Hospital during the last ten years, not a single case of acute perforation of peptic ulcer was found occurring in a female. Review of the literature on this subject shows males predominating by a striking majority, usually the ratio being about 10:1, but in no other considerable series was the female sex altogether excluded.

Incidence by Years.—Of the total 74 cases, distribution by years is shown in Table II.

TABLE II

INCIDENCE BY YEARS

1926	11
1927	11
1928	14
1929	13
1930	9
1931	11
1932	5
Total	74

There is seen to be a distinct decrease during the latter three years of this study. The explanation of this drop in numbers is not obvious, since the total hospital census during this period showed no proportionate change. It is suggested that increased unemployment since 1929 with less hurry and physical tension on those engaged at their occupations may be factors of some importance in the lessened incidence of ulcer perforation. It may be also that the public as a whole is becoming more "stomach conscious" in view of cancer campaigns of recent years, with the result that stomach lesions are being recognized earlier and are less frequently neglected to the point of perforation. That this latter is not the full answer, however, is evidenced by the fact that a few cases were encountered in the present series where perforation occurred in persons at the time actually under some dietary regimen for known ulcer. This finding is in accord with the experiences of Hinton[1] who reported six cases of acute perforation which occurred in patients under active treatment for chronic ulcer.

Seasonal Incidence.—Tabulation of cases by months disclosed no seasonal incidence of perforation. They occurred more frequently in October and December, and were less in April, June and August, but on the whole there was no significant variation between the months.

Age Incidence.—The youngest patient in this series was 19; the oldest 67,

there being two cases of this age, both of these incidentally being fatal cases. By decades, the 30–40 age period showed by far the largest number, the 40–50 period being next in frequency (Table III).

TABLE III

AGE INCIDENCE

10–20	1
20–30	16
30–40	26
40–50	17
50–60	10
60–70	4
Total	74

Location of Ulcer.—Contrary to our general impression prior to this review, the majority of the acute perforations were found in gastric ulcers, 41 being situated proximal to the pylorus. There were 26 lesions in the duodenum and seven were found at the pylorus. No multiple perforations were encountered. All the duodenal perforations in this series were on the anterior surface or superior border of the intestine. Of the stomach perforations, four were through its posterior wall into the lesser cavity. In these cases, as a rule, development of symptoms was later and the diagnoses more difficult. The remainder of the stomach lesions were about equally divided between the lesser curvature and the anterior surface. There was none noted on the greater curvature.

Previous Gastric History.—Not many years ago it was generally held that most peptic ulcers which resulted in perforation were acute ulcers, differing in type from the chronic peptic ulcer, being of relatively recent origin and preceded rarely by continued dyspeptic symptoms. More recently this opinion has been questioned though it still appears to have its adherents. In our series only 12 of the 74 patients gave a negative past gastric history, the remaining 62 or 86 per cent admitting more or less typical ulcer symptoms, varying in time from a few weeks to many years. These histories corresponded well with the operative findings. In only eight cases was there reported to be no, or slight, induration about the perforation as seen at the operating table. Eleven, or slightly over 15 per cent, of all the cases gave histories of previous nonoperative ulcer treatment. Two cases had had earlier acute perforations, one of these two having perforated twice previously (Table IV).

TABLE IV

HISTORY OF PREVIOUS GASTRIC COMPLAINT

Total No. Cases	Cases with Previous Gastric History	Cases Having Previous Nonoperative Ulcer Treatment	Cases with Previous Perforation	Operative Finding of Induration
74	62 (86 per cent)	11 (15 per cent)	2	58 (78 per cent)

Diagnosis.—The matter of diagnosis presented relatively little difficulty in this series. In 63 of the 74 cases the clinical picture was typical and the diagnosis was readily established. Of the remaining 11 cases the possibility of acute appendicitis was entertained in four cases, acute cholecystitis in three and acute pancreatitis in one. Since all of these eight cases were recognized and regarded as acute surgical emergencies, the possibility of perforated ulcer being considered in each, early operation was performed. The remaining three cases offered more difficult diagnostic problems. Each was admitted first to the medical service, two with the preliminary diagnosis of coronary artery disease and the third as intestinal neoplasm with partial obstruction. In each of these cases there was a delay of 24 hours or more before operative intervention, and all three proved eventually to be fatal cases.

Preoperative flat roentgenologic study as a diagnostic aid has proven of indifferent value to us. Flat abdominal films were taken in 32 cases and in 10 free gas was demonstrated; the remaining 22 gave negative results. Definite roentgenographic indication of right diaphragmatic elevation by gas is a most reliable diagnostic aid, but negative roentgenographic findings occur frequently and must be disregarded.

Operation.—The 74 operations were performed by nine different surgeons. All were simple closures, usually with purse-string suture, although a few were by mattress or figure of eight sutures. As a rule there were three suture layers; the first of catgut through all walls of the viscus, the second and third of linen not including the mucosa. An omental tab was usually included in the third suture layer. In three cases only a single suture layer was employed, one of these cases resulting fatally. Only six of the 74 cases were drained. In many instances there appeared to be great distortion and probable pyloric constriction after the plication closure. However, side-tracking or plastic procedures to obviate possible pyloric obstruction were deliberately withheld and in no case did interference with patency of the pylorus develop. Our experience shows that most of the apparent deformity disappears as the local inflammatory edema subsides.

General anesthesia was used more frequently than spinal in the total series, the ratio being 50 to 23. In the latter years, however, spinal and general anesthesia were about equally popular. One case was performed under local abdominal wall infiltration with novocain.

Immediate Results—Operative Mortality.—There were eight deaths in this series of 74 cases, an operative mortality of 10.8 per cent. No death occurred in a patient under the 30–40 year age period, the highest relative mortality being found in the 50–60 and 60–70 decades. Anatomic location of the lesion was not important, there being fairly equal distribution among the fatal cases: three gastric, three duodenal, and two pyloric. The type of anesthetic also seemed a relatively unimportant factor in mortality. There were deaths in three cases operated upon under general, four under spinal and one with local novocain, the latter in a very poor operative risk.

As was expected, we found that the patient's chances of surviving depended chiefly upon how soon after perforation his operation was begun. This relationship is shown strikingly in Table V.

TABLE V

MORTALITY AND ITS RELATION TO TIME OF OPERATION

Duration of Perforation	No. of Cases	No. of Deaths	Mortality Per Cent
6 hrs.	49	1	2
6–12 hrs.	13	2	15
12–48 hrs.	10	3	30
Over 48 hrs.	2	2	100
All cases	74	8	10.8

Experiences of Other Authors.—An extensive literature is to be found on the study of the general subject of acute peptic ulcer perforation. No attempt will be made, however, to present at this time findings of other authors except as they deal, at least indirectly, with late results. Although generalizations and impressions are found frequently expressed, studies with follow-up findings based upon direct examination and presenting criteria for analysis and specific findings are relatively few.

As is commonly seen on clinical investigation of similar subjects by different authors, no great unanimity of findings or conclusions exists in this field. This divergence of opinion may be explainable in part on the basis of local racial, geographic and dietetic variations. The type of primary operative procedure may also be a factor. But it seems most likely that lack of uniformity in evaluating results is the most important reason for this diversity of findings.

As regards the type of operative procedure to be employed for acute perforation, simple closure usually without peritoneal drainage seems now to be the method of choice in this country (Gibson,[2] Stewart and Barber,[3] Colp,[4] White and Patterson,[5] Pool and Dineen,[6] St. John,[7] Farr[8]). The British authors also, in recent years, have been favoring plication closure alone (Gilmour and Saint,[9] Bryce,[10] Black[11]). Urrutia[12] almost alone of continental European surgeons has been an advocate of this method. On the other hand, Deaver[13] urged the necessity of the addition of primary gastrojejunostomy to the closure, and this procedure is followed by many surgeons here and abroad (Walton,[14] Alexander,[15] Mills[16] H. P. Brown[17]). Still other writers have advocated primary anastomosis in most cases, though not as a routine (Scotson,[18] Shelley,[19] Platou,[20] Boyd[21]). In perforation of duodenal ulcers Moynihan[22] adds gastro-enterostomy to suture closure. For lesions situated at, or just proximal to, the pylorus he recommends excision of the ulcer with added pyloroplasty. So also does Söderland[23] who urges excision of the ulcer margins before suture closure in all cases. Pyloroplasty is advised by Hinton,[24] and H. P. Brown[25] in cases where primary gastro-enterostomy is not done. Kuntz[26] in some cases combines jejunostomy with simple closure, although he performs gastric resections in most instances.

In recent years more radical immediate measures for the treatment of acute ulcer perforation have been employed by central European surgeons. Particularly in the large German and Austrian clinics, primary gastric resection for perforation is now widely practised. Recently Graves[27] compiled a large series of such cases operated upon by several German surgeons, his findings indicating a lower immediate mortality rate with resection than with simple closure alone. However, in interpreting these results it must be realized that in this series the simple procedure usually was reserved for patients who were obviously bad operative risks. Many other reports of primary resections are to be found from foreign sources, some of the more recent of these being by Hromada and Newman,[28] Schwarz,[29] Kreuter,[30] Eichelter,[31] Judine,[32] Peters,[33] Ciancarelli,[34] and Kuhlman.[35] In this country gastric resection for acute perforation is rarely advised. The author recently performed such resection in a patient with multiple perforations with good result, and believes this procedure is indicated in some cases.

Considering now the question of published late results alone, reports as found in the literature show a wide divergence of findings and opinion. Thus Stewart and Barber[3] after simple closure reported their patients "in most instances cured or improved" and Walton[14] found "not a single recurrence" of symptoms in those who survived operation when primary gastro-enterostomy was performed. Gibson,[2] in a study of 93 patients, followed after operation, had 41 "excellent" and 31 "satisfactory" results. However, it is found on analysis of his statistics that, of the remaining 21 cases, 19 had symptoms sufficiently severe to require reoperation and of those reported as having good results two later had reperforation. Dineen[36] found 80 per cent of his cases to have good results and in an earlier study with Pool[6] reported 60 per cent to be satisfactory after simple suture alone. Employing the same operative method, White and Patterson[5] had 60 to 65 per cent "cure" of ulcer symptoms in a small series of cases. St. John[7] also reported favorable late results, finding 59 per cent symptom-free, and Farr[8] in a larger series found nearly 70 per cent good results after plication closure of acute perforation. Collinson[37] reported 65 per cent good late results. Southam[38] found 28 out of 37 cases to be satisfactory. On late investigation of 59 cases in which some had gastro-enterostomy in addition to simple suture, Shelley[19] found recurrent symptoms in only 32 per cent. K. P. Brown[39] states that over 60 per cent of his patients had no further trouble, the percentage of favorable cases being even higher in those who had primary gastro-enterostomy. Similarly Platou's[20] experience was favorable when he employed anastomosis at the time of operation for the perforation, all 14 of his patients so treated having had no further trouble. In reporting a group of 90 compiled cases, all of whom had primary gastro-enterostomy, Luff[40] states that over 68 per cent had "no pain or discomfort." These cases were not personally examined by him; also he does not give the time interval of the follow-up. Similarly vague is the report of Rhodes and Collins,[41] who found that 85 per cent of patients followed "reported themselves either entirely free of ulcer

symptoms, or evidences of recurrences were promptly controlled by dietary measures." Again there is no statement of the postoperative interval nor criteria used for evaluation of results.

In contrast to the above reviewed favorable reports are the experiences of other writers who found recurrent ulcer symptoms in many of their patients who had recovered from operative procedures directed towards the treatment of acute perforation. Lewisohn,[42] through personal late examination, in 33 cases found 13 "failures," five of these subsequently coming to reoperation. Williams and Walsh[43] reported that of eight patients with gastric ulcer perforation on whom late information was obtained, five had come to reoperation; of their duodenal cases 53 out of 94 had required further surgical treatment. Gilmour and Saint[9] in their analysis of 48 cases with late follow-up report 61 per cent recurrence of ulcer symptoms. In Platou's[20] series of 25 patients, after simple closure alone, 16 had recurrent ulcers. Of these 16, eight later had gastro-enterostomy performed and three others had reperforation. Morley[44] found that 17 out of his 41 cases, studied late, required further treatment for ulcer. Similarly Bryce[10] in 100 cases had only 32 free of gastric complaints. These experiences correspond to those of Scotson[45] who reports recurrent symptoms in 58 per cent of 92 cases. Similar also are the findings of Urrutia.[12] After late study this author reports recurrence of ulcer in 50 per cent of the cases treated by simple closure. After added gastro-enterostomy he found even more recurrences, only one out of three so treated being cured of ulcer in his experience.

Criteria for Evaluation of Late Results.—In dealing with patients presenting a gastric disorder, the accurate evaluation of late results is by no means easy. We must depend for our information primarily upon the patient's replies to questions about his subjective state. Tangible physical findings are few. Occasionally local tenderness may be found but its absence does not rule against the persistence of active pathology. Late roentgenographic studies were obtained in a few cases but it was not felt that sufficient information could be obtained in this way to warrant its being employed in the entire series. The expected postoperative deformity was seen in those cases so studied, but differentiation between active ulcer and cicatricial deformity particularly in the pyloric region could not be made with assurance. Gastric analyses also were not generally employed, since it was felt that this procedure would not materially aid us in evaluating results. In the main we had to depend primarily on the histories we could obtain.

We have then no yard stick by which to measure accurately the status of these patients. With criteria for recurrence or cure based largely upon subjective standards one cannot hope to grade results except in a gross way. Conclusions in any such study, if the attempt is made to tabulate results too finely, are liable to be misleading or unfair, no matter how earnest or sincere is the investigator.

We decided, therefore, to classify our postoperative results in a rough way, as good, bad or fair. In judging and gauging replies to our queries,

an attempt was made to evaluate in so far as possible the types of personalities with which we were dealing, to size up each individual and weigh his responses fairly. We have taken some time to clarify our standards, which we regard of prime importance in this type of study. Differences by various authors in criteria for cure and recurrence have caused much confusion in the literature.

Results classified as "good" were those in which patients had remained symptom-free after a period of reasonable dietetic and hygienic care. Such persons were expected to have returned to their usual occupations, or to have been able to do so, if the desire or opportunity were present. Preoperative weight and strength should have been regained and retained.

We regarded as "poor" results those cases which reported periodic or persistent recurrence of ulcer symptoms where these symptoms appeared real in origin and significant in amount. Any who required further medical or or surgical treatment for peptic ulcer were given this classification. Patients who had been reoperated upon were of course placed in this category. Cases with roentgenologic findings of new lesions or disturbances of gastric motility were put in the "poor" column.

In our final classification of "fair" results were included those patients whose complaints were mild, inconstant and not entirely typical of ulcer. Where symptoms were persistent, but appeared to be in large part functional or based on sociologic or economic dissatisfaction, the result was considered fair. Those individuals with recurrent complaints who admitted ignoring or seemed to have disregarded postoperative instructions soon after leaving the hospital were regarded as having fair results unless their symptoms were of more than moderate severity.

Analysis of Late Findings and Results.—By these standards, of the 32 who had recent follow-up examination, 15 were classified as having poor results, six as fair and nine as good. Recurrence of symptoms in this group was found in 23 of the 32, or 71.7 per cent. Similar evaluation was attempted in those heard from by questionnaire only. Of these 13 cases the results in five were considered poor, one fair and seven good, giving six or 46 per cent with recurrence of symptoms. Altogether of the 45 cases about whom we had recent information, 29 were found to have had significant further gastric trouble, a recurrence in the series of 64.4 per cent.

It is interesting to note that of these 45 cases who were followed over a one to six year period after perforation, five have had subsequent reoperation, three for a second acute perforation (the third perforation for one patient). One had a gastro-enterostomy six months postoperative for obstruction and one a gastric resection for persistent symptoms. This one case with gastro-enterostomy was the only one we know of in our entire series in whom pyloric obstructive symptoms developed after plication closure alone for acute perforation, this despite frequent gross distortion of the pylorus immediately after the closure.

TABLE VI

ANALYSIS OF END-RESULTS

	Number of Cases	Poor	Fair	Good	Total Recurrences
Special examination	26	12	6	8	23 (71.7 per cent)
Recent follow-up examination	6	3	2	1	
Questionnaire cases	13	5	1	7	6 (46 per cent)
Total examinations plus questionnaire cases	45	20	9	16	29 (64.4 per cent)

SUMMARY.—(1) A study is presented of 74 consecutive cases of acute perforation of peptic ulcer treated by simple closure alone.

(2) These cases were all from the Surgical Services of the Beekman Street Hospital, operated upon during the years 1926–1932, inclusive.

(3) It is interesting to note that all of these patients were males, and that during the last ten years not a single female has been admitted to the Beekman Street Hospital with this complication.

(4) During the latter three years embraced by this study, there has been noted a rather sharp drop in the incidence of ulcer perforation. The cause of this decrease is not clear; several possible factors are considered.

(5) No significant monthly or seasonal variation was found.

(6) By far the largest number occurred in the 30–40 year age period.

(7) Gastric perforations were more frequent in this group than were duodenal or pyloric, the anatomic distribution being gastric 41, duodenal 26, pyloric seven.

(8) Of the 74 patients, only 12 had been entirely free of "dyspeptic" symptoms prior to perforation. The remaining 62 or 86 per cent gave a definite antecedent gastric history. Eleven of these had had previous medical ulcer treatment with two having had earlier simple closure for acute perforation. One patient had perforated twice previously.

(9) The majority of cases were seen early after onset of acute symptoms, 49 or 65 per cent having been operated upon within six hours from the estimated time at which perforation occurred.

(10) On the whole diagnosis was made easily; 63 of the cases presented no diagnostic problem.

(11) Flat abdominal roentgenologic study proved to be only an indifferent diagnostic aid. It is valuable only when free subphrenic gas can be demonstrated. A negative finding has little significance.

(12) Operation consisted almost invariably of three layer purse-string closure of the perforation. Peritoneal drainage was rarely employed.

(13) Eight of the 74 patients died after operation, a general operative mortality of 10.8 per cent. Neither anatomic site of the perforation nor choice of anesthetic was found to be a factor of consequence in mortality in this series.

(14) Mortality percentage rose directly and sharply with delay in commencing operative interference.

(15) The literature dealing with late results in acute ulcer perforation is reviewed.

(16) The author's criteria for evaluating late results are presented in detail.

(17) Through combined special examination and replies to questionnaire, late study was obtained in 45 of the total 66 patients who survived operation, giving recent information in 68.1 per cent of these cases. The follow-up period was over a one to six-year period.

(18) Altogether of the 45 cases, about whom we had recent information, 29 were found to have had significant further gastric trouble, a recurrence in this series of 64.4 per cent.

CONCLUSIONS

(1) The low mortality rate and excellent early results, with simple closure alone, warrant the routine use of this procedure in the emergency treatment of acute peptic ulcer perforation.

(2) Addition of gastro-enterostomy for mechanical reasons is rarely necessary, no matter how extensive the induration or how great the apparent pyloric distortion after plication.

(3) Acute perforation followed by successful closure results in permanent cure of ulcer in only a minority of cases.

(4) In almost two out of three cases peptic ulcer symptoms recur later with complaints of greater or lesser severity.

(5) All cases of ulcer perforation deserve postoperative follow-up study and in most instances further medical or surgical care is required.

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OBSTRUCTIONS ABOUT THE MESENTERY IN INFANTS*

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REPEATED vomiting in infancy is always a cause of great concern to the parents and to the attending physician. When the baby cannot retain his feedings in the first weeks of life the usual procedure is to try various modifications in the diet. If after several failures, and the response even to thickened feedings and atropine therapy is without result, an organic obstruction of some type is likely to be found the underlying cause. The most common etiologic factor of organic obstruction in infancy is hypertrophic pyloric stenosis. But there are a certain number of obstructions which make their presence known almost as soon as the baby takes anything by mouth. These obstructions can be differentiated from hypertrophic pyloric stenosis. They occur in or about the duodenum and are usually in close anatomic relationship to the mesentery of the small intestine. We wish to consider this group of obstructions in this communication. The group includes the duodenal atresias; the internal herniae; and the anomalies due to faults in migration, descent and fixation acting on the first portions of the small intestine.

The earliest recorded case of congenital intestinal occlusion is credited to Calder who, in 1733, according to Cordes[1]† described an atresia of the duodenum. In 1901, Cordes collected from the literature only 56 cases of duodenal atresia and stenosis. Two years later Kuliga[2] made an extensive study of congenital occlusions of the entire intestinal tract based on 189 reported cases. In 1922, Davis and Poynter[3] increased this number to 392. In other words, the reported cases averaged two per year over a period of approximately 200 years, which gives a false impression of the frequency with which the condition occurs. While congenital intestinal anomalies leading to complete and partial obstruction are unusual, they are not rare. Ladd,[4, 5] who reported 60 cases from the Children's Hospital of Boston, states that the fact that he has "encountered ten cases in a comparatively short time makes me feel that they (congenital anomalies) have been overlooked in the past." He feels that the "frequency of postmortem reports of the cases and the infrequency of these conditions in adult life indicate a high mortality without surgical intervention."

Since Cordes' contribution other reports stressing various phases of congenital occlusions have appeared. Among these should be mentioned those of Pehu and Auberge,[6] Thorndike,[13] DeSanctis and Craig[7] and most re-

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† 1752 according to Davis and Poynter.[3]

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cently Ladd. Spriggs[8] gives an excellent and detailed description of the microscopic findings in cases of stenoses, as do Davis and Poynter. The latter feel that the fundamental etiologic factor is primary sclerosis of the regional vessels, the changes in the bowel wall being secondary to the resulting circulatory disturbances.

Congenital intestinal occlusions are commonly classified as intrinsic and extrinsic. The pathology of the atresias varies from a complete absence of a portion or portions of the intestine to all grades of intestinal fibrosis and diaphragms occluding the lumen. Atresias occur more commonly in the duodenum than in any other part of the gastro-intestinal tract, 30 per cent according to von Koos[9] and Davis and Poynter. The extrinsic occlusions form an interesting group and it may be said that in general they result from faulty or incomplete intestinal rotation and hyperperitonealization during fetal development. Internal herniae as a cause of obstruction are encountered less frequently than short mesenteric arteries, incomplete intestinal rotation, abnormal peritoneal bands and adhesions.

An understanding of the embryologic development with the changes in anatomic relationships of the gastro-intestinal tract will help the surgeon to untangle these complicated anomalies which he encounters at operation. A fairly accurate picture of the process has been pieced together for us by studies on human embryos and from observations in comparative anatomy.[18] It is presented as a theoretical motion picture although the evidence is made up from a series of still exposures often with great unfilled gaps between. When one realizes the many opportunities that there are for something to upset the orderly progression it is amazing that these anomalies are so relatively uncommon.

The stomach moves down from the cervical into the lower thoracic region during the sixth and seventh week of embryonic life. It hangs vertically with a right and a left surface until a more rapid growth of the dorsal or posterior border and its mesentery takes place. This inequality in growth rate leads to the development of a relatively freely movable greater curvature and a relatively fixed lesser curvature. The fundus also evolves as an outpouching on this dorsal border. The resulting disproportion between the anterior and posterior border of the stomach leads to a rotation, the greater curvature moving towards the left. The former right surface finally becomes posterior and the left surface anterior in position. During this same time the duodenum is giving rise to the buds which develop into the liver, biliary passages, and the pancreas. The duodenum then becomes occluded by proliferation of its lining epithelium. It remains in this condition so that the lumen is blocked until the sixth week. Then, normally, the epithelium is absorbed again. In rare instances this does not occur but the epithelium becomes organized. This mechanism results in the various types of atresia encountered in the duodenum as well as elsewhere in the jejunum and ileum. These atresias may be single or multiple, partial or complete. The occlusion may be a solid cord of some extent or a simple membrane. Cases 1 and 2 illustrate this type of anomaly. Atresia in the duodenum frequently occurs

just above the opening of the common duct, but we have not met such a case in our series.

The mobile duodenum undergoes a rotation and fixation of its own from the sixth to the ninth week. It is forced towards the right side of the abdomen by the rotation of the stomach, and there twists on its axis. This causes an alteration in the position of the common duct and the pancreatic outgrowths so that they occupy more nearly their adult positions. The duodenum then becomes fixed to the region of the gallbladder and liver, fused with the dorsal wall, and suspended behind the root of the mesentery by a fibromuscular band, Treitz's ligament. The superior mesenteric artery thus passes in front of the terminal part of the duodenum.

That portion of the intestinal tract supplied by the superior mesenteric artery—the midgut—starts out as a U loop within the umbilical cord. In the sixth week of life the cecal bud becomes apparent. By the ninth week coils of lower jejunum and ileum have formed in the cord and an upper jejunal coil distal to the duodenojejunal attachment. The loops in the cord are drawn into the abdominal cavity by retraction bands in the mesentery during the tenth week. The operation of rotation of the bowel which is necessary before its fixation takes place appears to be greatly helped by its shape and position in this special umbilical sac. The portion of the midgut from the duodenum to the vitelline artery passes in behind the artery from right to left. The portion of the midgut from the vitelline artery to the midtransverse colon passes in front of the artery from left to right. By the third month the U shaped loop has become twisted around the axis of the superior mesenteric artery.

The hindgut is drawn up and molded into place by pressure of the small bowel against it. It usually fuses with the posterior wall assuring its fixation. Towards the end of the third month the large bowel occupies an oblique position across the abdomen from the right iliac crest to the left hypochondrium. The rotation has brought the mesentery of the cecum and ascending colon into contact with the duodenum. The cecum thus comes to rest in front of the lower part of the right kidney near the gallbladder in the majority of fetuses. It remains there until about the time of birth. At the time of birth the ascending colon moves toward the right iliac fossa, elongating in the process—descent. The liver and the gallbladder move upwards providing more room for fixation of the ascending colon at the hepatic flexure. The adhesions which the ascending colon forms in its fixation, just before and just after birth as the cecum descends to its final position, offer many opportunities to produce anomalies.

If there are unequal tension pulls on the retroperitoneal duodenum it may be angulated or twisted on itself, especially since it may be caught between two opposing forces—those causing its firm anchorage at Treitz's ligament and those pulling towards the right iliac fossa. Under these conditions it is not strange that we have encountered such partial obstructions as are recorded in Cases 5, 6, 7, 8, and 9. These result from hyperfixation

of the peritoneum about the duodenum giving partial obstructions from plications or torsions of its lumen. In Cases 10 and 11 we have examples of obstructions due to failure in descent of the cecum. These patients presented an exact duplication of the position and attachments of the great bowel at the end of the third month of development. But their close attachments prevented the duodenum from functioning properly, especially in Case 11 where a well formed cystocolic membrane was exposed. In Case 4 we have a most interesting example of one of these developmental anomalies. It is classed as a retroperitoneal hernia by most surgeons. We believe that it represents an abnormal fixation of the mesentery. When the rotation occurs in a direction opposite to the normal, the duodenum and mesentery come to lie in front of the transverse colon instead of behind it. These cases are a great puzzle to the surgeon unless he can expose the root of the mesentery. Then, the whole bowel unrolls rapidly. At the finish of such an operation all the small bowel will occupy the right and all the large bowel the left side of the abdominal cavity. This is the condition as it is in development before any rotation has taken place.

When there is failure of complete fusion at the duodenojejunal junction, a deep fossa may be left into which jejunum may herniate. Case 3 is an example of a very unusual herniation into the lesser peritoneal cavity through such an opening.

Herniation of the small intestine into the lesser peritoneal cavity is uncommon. Moynihan,[10] in his monograph on retroperitoneal hernia, cites only four cases. The bowel gains access to the lesser peritoneal cavity by two routes, the more common being through the foramen of Winslow. Dewis and Miller[11] reported 34 cases. Less frequently as in the present case, herniation occurs through the transverse mesocolon. Heaney and Simpson[12] reported 21 cases of this type. The herniated intestine may remain in the lesser peritoneal cavity or it may partially emerge by forcing itself through the gastrohepatic or gastrocolic omentum to again occupy, more or less, its normal position in the abdominal cavity. Occasionally herniae through the transverse mesocolon emerge through the foramen of Winslow.

In reported cases increased intra-abdominal pressure due to vomiting caused by associated lesions (ulcer or carcinoma of duodenum and stomach) was felt to be responsible for producing a tear in the transverse mesocolon. Its occurrence in adult life makes it probable that in most instances it is an acquired lesion. There seems to be little question that in the present case the defect in the mesocolon was congenital.

CASE REPORTS

Case 1.—No. 34798. E. S., a white female infant, the fourth in the family, was born in the Strong Memorial Hospital, May 1, 1930. The father and mother were healthy with negative Wassermanns. The pregnancy had been normal, the delivery spontaneous, and at birth the child was apparently normal weighing 2,600 Gm. (5.7 lbs.). The patient was seen in consultation by the pediatricians on May 3 because of vomiting and failure to pass urine and meconium. Vomiting started 48 hours after birth. Regurgitation

was frequent, occurred immediately after feeding and on some occasions consisted of a thin brownish fluid containing "coffee ground" material.

Physical examination showed a dehydrated female who appeared normal. Abnormal findings were limited to the abdomen which was thin walled and distended in the epigastrium but scaphoid in the lower abdomen. A sausage shaped mass extended diagonally across the epigastrium from the left costal margin to the right flank about on a level with the umbilicus. Across this mass peristaltic waves were visible, passing usually from left to right but occasionally from right to left. The mass was soft and doughy. The liver edge was palpable 1 cm. below the costal margin. No other masses were palpable. Bladder dulness was not increased to percussion.

Frequently repeated subcutaneous saline infusions were given. A catheter introduced into the rectum passed readily without encountering an obstruction. A small amount of normal saline which was introduced was readily expelled and with it a thick tenacious plug or cord of meconium 10 cm. in length. Following this more meconium was passed. The size of the abdominal mass remained unchanged. On May 4, the third day, the signs and symptoms were unchanged. A barium enema showed the colon patent and filled up to the hepatic flexure. At this point there was a "redundant loop of bowel distended with gas." The barium did not fill the ascending colon or cecum. The patient was first seen by the surgeons May 5, the fourth day, at which time her condition was grave. She was almost moribund. Her weight was 2,260 Gm. (4.95 lbs.), a loss of 340 Gm. (.74 lbs.). The temperature was 35° C. The abdominal signs were unchanged.

Operation was performed immediately under ether anesthesia, a right rectus incision being made. The stomach and duodenum were tremendously distended, the diameter of the latter being about 8 cm. and its walls of tissue paper thinness. The duodenum was bound down by abnormal peritoneal bands which were released. The transverse colon and stomach were reflected upward and Treitz's ligament identified. The jejunum appeared normal though small, being about the size of an ordinary lead pencil. Dilatation of the duodenum was found to extend to the third portion at the point where the superior mesenteric vessels crossed, these having been reflected upward and any possible constricting effect there relieved. Gentle pressure was exerted on the dilated duodenum but no gas passed beyond the point of obstruction. It was thus apparent that the obstruction was intrinsic in the retroperitoneal portion of the duodenum and due either to an occluding membrane or an atresia. Relief of the obstruction depended upon a short circuiting anastomosis. Duodenojejunostomy, the logical procedure, was considered inadvisable and almost impossible due to the thinness and delicacy of the duodenal wall as a result of distention. It was questionable if the thinned out walls would hold the sutures. A posterior gastro-enterostomy was performed without clamps, using silk sutures throughout. The stoma was approximately 4 to 5 cm. The patient's condition at the end of the operation was poor.

The immediate postoperative recovery was excellent. The temperature rose to 39° C. the day after operation, returning to normal on the third day. Subcutaneous saline infusions were given regularly. Feedings were started the day after operation. The patient vomited once and thereafter feedings were given by gavage; there was no further vomiting. She took the bottle on the third day after operation and from then on progressed satisfactorily. She regained all weight lost and when discharged 15 days after operation weighed 2,620 Gm. (5.7 lbs.). A normal stool was passed on the second day after operation. Urinary function became normal. A gastro-intestinal series two days before discharge showed a dilated stomach and a well functioning gastro-enterostomy (Fig. 1).

Unfortunately, it was soon recognized that the patient was a Mongolian idiot. Her course from the gastro-intestinal standpoint was entirely satisfactory for one year. Irregular attacks of vomiting and diarrhea occurred about a year after operation. These

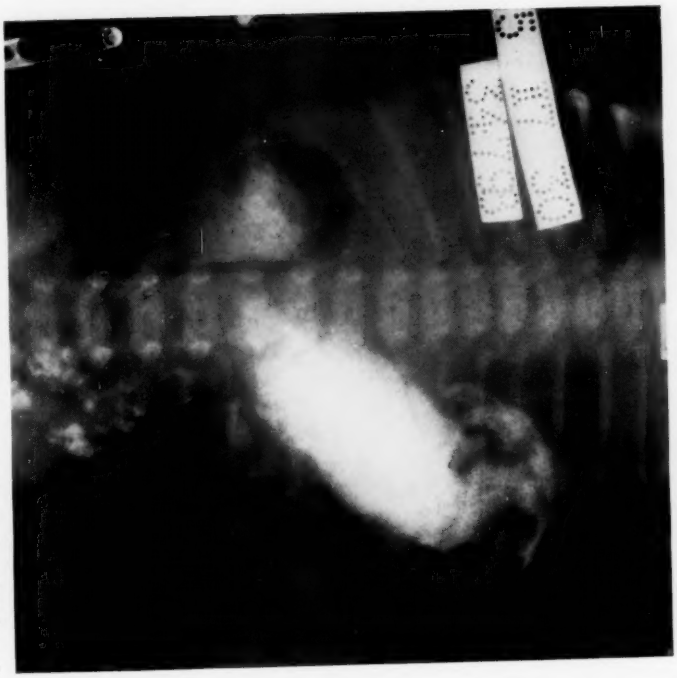

FIG. 1.—Case 1. Roentgenogram showing a dilated atonic stomach and duodenum. Taken after gastro-enterostomy.

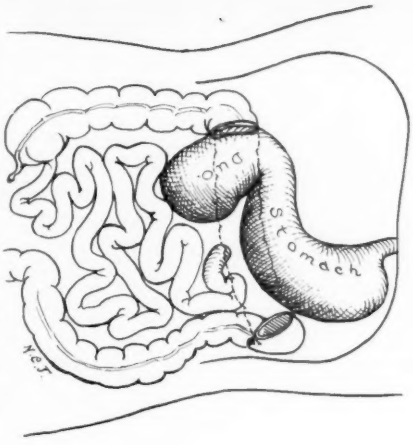

FIG. 1a.—Case 1. Atresia of duodenum. Condition found at operation. Absence of third part of duodenum. Confirmed at autopsy two years later.

persisted for a period of one or two months and then ceased. In January, 1932, 20 months after operation, she was readmitted to the hospital with a diagnosis of pneumonia, to which she succumbed.

Postmortem examination showed bilateral bronchopneumonia, acute bronchitis, bilateral otitis media, acute splenic tumor, atresia of the duodenum with complete absence of the third portion, a healed, patent gastro-enterostomy and a patent foramen ovale.

Case 2.—No. 61269. J. L., an apparently normal female, the fourth child, born in the Strong Memorial Hospital, May 22, 1932, began vomiting on the fifth day. Changes in formula, antispasmodics, *etc.*, failed to relieve symptoms. During the first 11 days of life the weight decreased from 3,350 Gm. (7.37 lbs.) to 2,620 Gm. (5.76 lbs.). Physical examination on June 3 was essentially negative except for visible peristaltic waves just below costal margins, traveling from left to right, and incomplete closure of the umbilicus. A small amount of barium administered the same day showed dilatation of the first portion of the duodenum with complete obstruction about the beginning of its second part. Adequate fluid intake had been maintained by administration of subcutaneous saline.

Operation was performed on June 4. A tremendously dilated duodenum presented itself. The transverse colon was small and occupied its normal position. It was reflected from its normal attachment allowing exposure of the retroperitoneal portion of the duodenum which was explored as far as Treitz's ligament. It was seen that a considerable portion of the ascending colon was also retroperitoneal and it was released from its retroperitoneal position. A clear view of the entire small intestine was thus possible and no obstruction was apparent at any point except where the duodenum entered its retroperitoneal position. The retroperitoneal duodenum was small and collapsed but appeared normal. The pancreas appeared normal. There were several small aberrant pancreatic nodules in the wall of the dilated duodenum. Pressure on the dilated duodenum failed to force any gas into the distal retroperitoneal duodenum. The duodenum was opened longitudinally at the point of obstruction exposing a diaphragm about 2 Mm. in thickness apparently completely blocking the lumen of the bowel. Bowel contents were carefully aspirated. A normal bowel lumen was readily demonstrable below the diaphragm. The diaphragm was completely removed by monopolar desiccation, which adequately controlled all hemorrhage. The intestinal lumen was restored by suturing the bowel transversely with silk and reenforcing with mattress sutures of the same material (Fig. 2).

While at no time during recovery was the patient's condition alarming, considerable difficulty was encountered. A 50 cc. transfusion was given following operation and numerous subcutaneous saline infusions during the first four days. Vomiting and distention were troublesome until the twelfth day postoperative. What feedings were retained were supplemented by saline infusions. Gastric lavage was used frequently. It was not until about two weeks after operation that the patient stopped losing weight and began to retain feedings with any regularity. Fluoroscopy showed slight delay in emptying of the stomach but free passage of barium along the upper intestinal tract. All vomiting finally ceased and the patient was discharged July 13, 1932, on the thirty-ninth postoperative day. The weight on discharge was 3,440 Gm. (7.48 lbs.).

The patient was readmitted on two or three occasions during the next few months for coryza, otitis media and on one occasion because of diarrhea and vomiting, neither of which was observed during a subsequent five day stay in the hospital. The general course has been one of constant improvement and normal development. A gastro-intestinal series July 6, 1932, a month after operation, showed some enlargement of the stomach and dilatation of the duodenum but no obstruction to the flow of barium. In December, 1932, at age of six months, she was observed in the hospital for a period of six days. She was admitted because of apparent abdominal pain and constipation. Thorough study showed no evidence of obstruction. In October, 1933, a year and five months after operation, she was readmitted because of intermittent vomiting over a

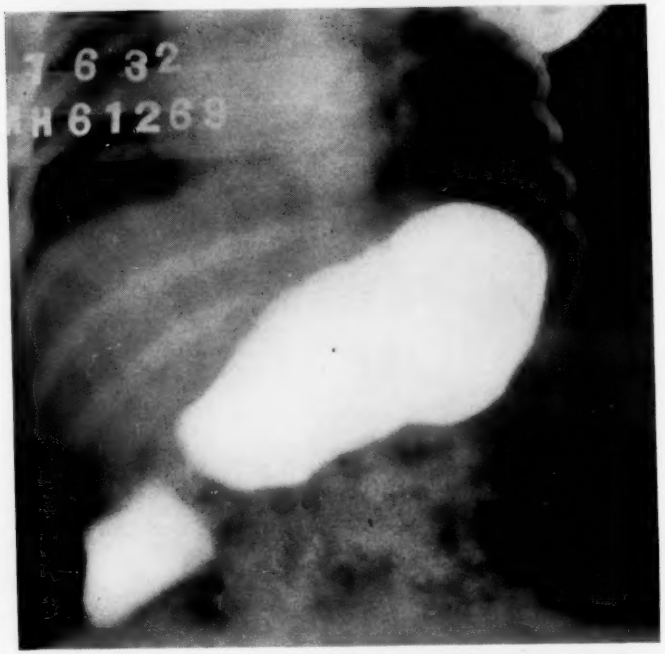

FIG. 2.—Case 2. Dilated atonic stomach and duodenum as shown by roentgenogram.

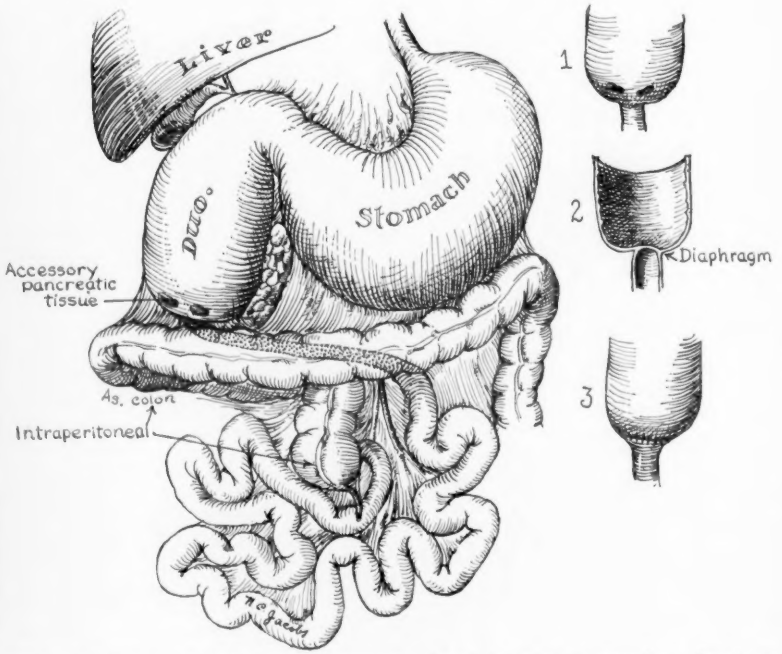

FIG. 2a.—Case 2. Atresia due to diaphragm. Condition found at operation. Repair as carried out to right indicated in Inserts 1, 2, and 3.

period of two months. A gastro-intestinal series at this time showed no definite obstruction. The stomach emptied itself slowly and passage of the barium through the intestinal tract was slow but no point of obstruction was noted. During 1934 there were no gastro-intestinal complaints. Illnesses during 1934 consisted of pyuria on two occasions and whooping cough, otitis media again in 1935, also measles and furunculosis.

Case 3.—No. 35271. E. W., white, male, the third child in the family, age three days, was referred to the Strong Memorial Hospital May 23, 1930, because of persistent vomiting since birth. The antecedent history was entirely negative, both parents being normal adults. The pregnancy and delivery had been uncomplicated. At birth the patient was apparently a normal healthy infant weighing nine pounds. Breast feedings had been started immediately. Vomiting, projectile in type, started immediately after the first feeding and had persisted until the time of admission to the hospital. It occurred regularly about 15 minutes after each feeding and apparently the entire feeding was vomited. The stools, at first greenish in color, became slightly yellow on the third day. There had been a slight discharge from the left eye since birth. On admission to the hospital the patient weighed seven pounds, 14 ounces and physical examination was entirely negative except for a slight conjunctivitis of the left eye. Examination of the blood presented no abnormalities in the cellular elements but the chemical analysis disclosed a non-protein nitrogen of 74 mg. per 100 cc., while the chlorides were 675 mg. per 100 cc. Stool examination was negative. Vomiting persisting despite treatment, a small amount of barium was given by tube the day after admission and its course followed by fluoroscopic examination. The esophagus, stomach and duodenal cap appeared normal except that the latter remained filled and very little if any of the barium progressed into the distal portions of the duodenum. At the end of an hour practically all the barium was still in the stomach, only traces being discernible in the jejunum, which showed vigorous peristalsis. The course of the barium was observed at intervals and at the end of 19 hours no barium remained in the small bowel, about one-quarter being in the ascending colon and the remainder being still in the stomach. It was quite apparent that the infant was suffering from a partial obstruction of the upper gastro-intestinal tract distal to the first portion of the duodenum, the exact cause being undetermined (Fig. 3). With the hope that the underlying cause was such that the obstruction might eventually relieve itself, operative treatment was delayed. During this observation period the infant was given various formulae without any improvement. Dehydration was combated by repeated subcutaneous injection of normal saline, 1,200 cc. administered over a three day period.

Operation was performed May 27 under ether anesthesia. On opening the abdominal cavity through a high right rectus incision, the duodenum, somewhat dilated, was found to occupy a high position. The stomach and pylorus appeared normal. Above the lesser curvature of the stomach in the lesser peritoneal cavity could be seen dilated loops of small bowel. The transverse colon and stomach were reflected upward and Treitz's ligament identified. The jejunum was carefully picked up and an attempt made by gentle traction to deliver it into the wound. There was a slight resistance which on being released, disclosed, in the region of Treitz's ligament, a dilated segment of jejunum which immediately exhibited vigorous peristalsis. At each end of this dilated portion of jejunum there were apparent, across the bowel, definite markings, indicative of constriction. The release of the obstructed segment and its appearance in the wound occurred so rapidly that the opening by which it gained access to the lesser peritoneal cavity was not identified. The intestinal loops had disappeared from the lesser peritoneal cavity and as a rapid examination of the remainder of the gastro-intestinal tract showed no other abnormality, the abdomen was closed.

The postoperative convalescence was entirely satisfactory. There was a moderate amount of vomiting during the first 48 hours during which time an adequate fluid intake was maintained by subcutaneous administration of normal saline solution. The subsequent course in the hospital was uneventful, the infant retaining the prescribed formula

without difficulty. At the time of discharge from the hospital June 3 the infant weighed 3,480 Gm. (7.65 lbs.). About two weeks after discharge there occurred a mild gastrointestinal upset which was readily controlled by a change of formula. On June 25, the patient weighed 4,450 Gm. (9.79 lbs.).

Case 4.—No. 56990. T. C., a white male, age two and one-half months, the sixth child, was admitted to the Strong Memorial Hospital January 29, 1932, because of convulsions. At birth the patient had appeared normal and weighed nine and one-quarter pounds. Projectile vomiting began immediately after birth and still persisted at the time of admission. It occurred usually soon after feeding. At times it would cease for as long as three to four days. Sometimes evening feedings were retained and vomiting would occur in the morning, the vomitus under such circumstances consisting

FIG. 3.—Case 3. The peculiar shadow noted along the lesser curvature is due to a loop of jejunum herniated into the lesser peritoneal cavity. The faint shadow back of the stomach indicates the position of this loop of bowel from its entrance in the paraduodenal fossa.

of the feeding of the previous evening. Occasionally it was yellow or green but generally it consisted of the food taken at previous feeding. The patient was breast fed for two weeks, following which various formulae were tried. Constipation was marked until shortly before admission. Three weeks before admission, while feeding, the patient suddenly became unconscious, remaining so for five to ten minutes. The evening before admission he had the first convulsion, which was repeated several times during the night. Following the first convulsion abdominal distention developed. This was relieved by an enema. There had been slight jaundice for the first two weeks of life.

Physical examination showed a well developed but undernourished male infant, lying with eyes open and eyeballs rolled upward and rotated to the right. The extremities were flexed in a clonic state, but could be extended. Fists were clenched, no cyanosis. While palpating the abdomen, the patient suddenly relaxed and the eyes straightened.

Respirations were irregular. Suddenly the left arm began to jerk, the hand was again clenched and the patient became unconscious. Respirations stopped, the heart beat which had been forceful became distant. Artificial respiration stimulated breathing but respirations continued to be shallow. Convulsions continued throughout the examination. The heart and lungs were normal. The abdomen was moderately distended with the liver edge palpable at the costal margin. The tendon reflexes could not be elicited. There was no clonus. Chostek and Babinski were negative. The temperature was normal.

Laboratory examinations showed: red blood cells, 3,600,000; hemoglobin, 60 per cent; white blood cells, 15,200. The platelets were slightly decreased. The differential was normal. The urine and stool were negative. Blood chemistry showed calcium 9.6 mg., phosphate 5.6 mg., chlorides 410 mg., CO_2 70 vols. per cent. The Wassermann was negative. Lumbar puncture showed a pressure of 15, clear fluid, Pandy negative and one cell.

During the first few days in the hospital the patient's temperature varied from normal to 39.5° C. Vomiting persisted with more or less regularity and was projectile in type. Examination of the vomitus failed to reveal any occult blood. The stools were watery. Five days after admission a left myringotomy yielded a moderate amount of pus. The fluid level was maintained by subcutaneous saline as indicated. Eight days after admission a transfusion was given. On February 16, 1932, a roentgenologic gastro-intestinal study showed some pylorospasm, but more important, a marked delay in emptying of the duodenal cap due to some obstruction in the duodenum just beyond the cap. The duodenum was found to be in an abnormal position. All possible variations of formulae and antispasmodics were tried in spite of which vomiting persisted, and even increased in severity and frequency. Despite the patient's poor condition from infection (ears), *etc.*, laparotomy was performed February 26.

Operation was started under local infiltration which was later supplemented with light ether. The stomach and duodenum were distended. Fine filmy peritoneal bands crossed the duodenum in several places. The omentum was attached to the duodenum. The common duct and pancreas were easily exposed and visualized. The duodenum was dilated to the point where the mesenteric artery and vein crossed. The latter were incorporated in a constricting band. There was no apparent exit of small bowel from this point. Loops of small bowel were visible retroperitoneally in the left side of the abdomen. Involved in the above mentioned constricting band were three loops of large bowel, part of which were adherent to the duodenum in a retroperitoneal position. The cecum and appendix, crossing on top of these loops, were securely fixed by additional bands (Fig. 4). Gentle traction on exposed large and small intestinal loops failed to release this complicated tangle in any way. The lateral attachment of exposed bowel was divided far over on the right side and down to the posterior abdominal wall. It then became apparent that the ascending colon and cecum had looped back around this lateral attachment. The transverse colon had become attached by a peritoneal band in front of the duodenum. By freeing the cecum and appendix it was possible to draw out from its retroperitoneal sac the entire small intestine which was collapsed and dark blue in color from obstruction of its circulation. By careful dissection down to the mesenteric vessels the transverse colon was freed, after which it was possible to untwist the small bowel, thus entirely relieving the obstruction. With the untwisting of the small bowel it immediately flushed to a bright pink color, indicating the release of the obstruction to the blood supply. The small bowel now occupied the entire right side of the abdomen and entered directly into the large bowel, which occupied the entire left side and was not attached in any way to the posterior abdominal wall. The whole gastro-intestinal tract was thus one continuous tube from the pylorus to the sigmoid. The opening in the small mesentery was closed with silk. The operation was surprisingly well tolerated.

The postoperative recovery was entirely satisfactory. Except for some vomiting

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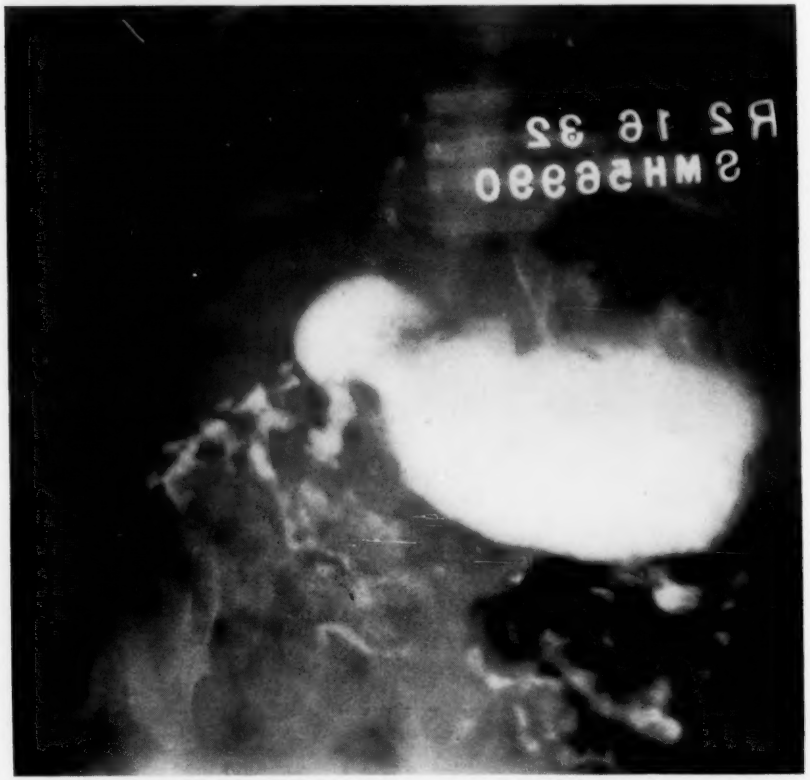

FIG. 4.—Case 4. Roentgenogram showing the characteristic dilated stomach and duodenum.

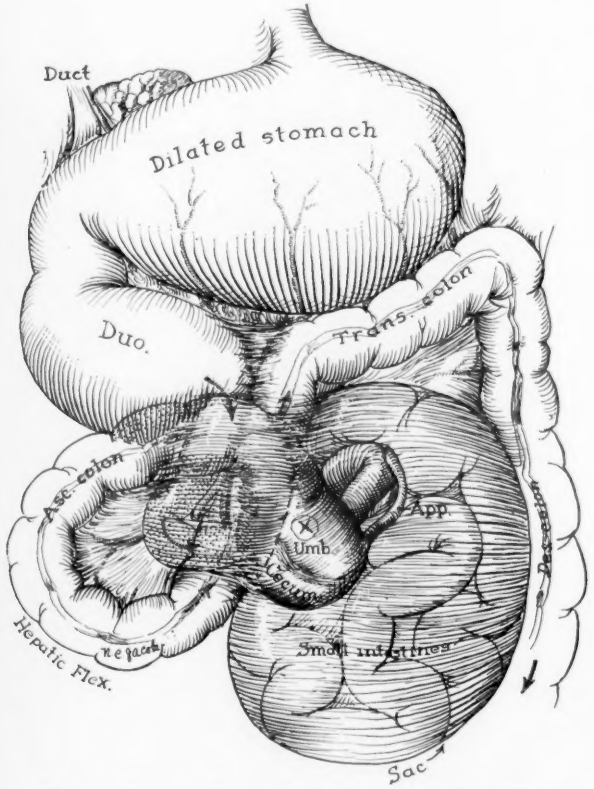

FIG. 4a.—Case 4. The condition found at operation. This is not an internal hernia but a reversed rotation of the midgut on the mesentery. This obstruction is easily handled by exposing the root of the mesentery and twisting the bowel in the reverse direction.

immediately following operation, feedings were well taken and retained. There was a slow steady gain in weight.

Case 5.—No. 107422. J. G., the fifth child in the family, was born prematurely in the Strong Memorial Hospital, September 26, 1935. She weighed 2,480 Gm. (5.45 lbs.) at birth. Vomiting (regurgitation) began on the third day and became projectile in type on the seventh day. It occurred immediately after feeding and consisted of the food previously ingested. Thrush which developed on the sixth day was successfully treated. No bowel movement from October 2 to October 7. An enema on October 7 effected the discharge of some greenish-brown fecal material. There was subsequently a normal stool which was guaiac positive. Changes in formula, antispasmodics, refeeding and gavage failed to control symptoms and the patient continued to lose weight. On October 7, 1935, at the age of 12 days, the weight was 2,300 Gm. (5.0 lbs.). Frequent saline infusions had been given.

Physical examination at this time showed a small, emaciated, premature infant with pinched facies. There was slight cyanosis of lips. The head appeared large in proportion to the size of the body and the anterior fontanelle was soft. There were white membranous patches on the inside of each cheek and on the tongue. The umbilicus was practically healed and there was slight herniation. Otherwise the abdomen was negative.

A gastro-intestinal series made on October 9 showed the stomach and upper intestinal loops filled with gas. The barium left the stomach very slowly.

Operation was performed October 11 under ether anesthesia through an upper right rectus incision. The stomach was large and dilated. The pylorus was normal. The duodenum was dilated and was attached throughout its entire length by peritoneal bands which ran across the duodenum and transverse colon, anchoring it in several kinks making an accordion pleated type of obstruction. The attachment of the transverse and ascending colon was freed to the cecum, after which the large bowel was rotated to the left and the adhesions between the duodenum and mesentery were carefully divided all the way to Treitz's ligament. This allowed the kinked portion of the duodenum to straighten out and assume a normal position. The abdomen was closed with silk.

The postoperative course was satisfactory although somewhat difficult. One transfusion was given postoperatively. Subcutaneous administration of saline and glucose was given repeatedly and regularly during the first five days following operation. Vomiting occurred irregularly during the first ten days, but there was none thereafter. Her general condition improved steadily and at the time of discharge on November 27, aged 62 days, her weight was 3,340 Gm. (7.3 lbs.).

The child was last seen March 2, 1936, aged five months, at which time she was symptom free and weighed 5,840 Gm. (12.8 lbs.), an increase of 3,440 Gm. (7.4 lbs.) since birth.

Case 6.—No. 105930. J. B., a white, female, first child, age 34 days, was admitted to Strong Memorial Hospital September 20, 1935, because of persistent vomiting. Vomiting began about the ninth or tenth day of life and at first occurred only once daily or on alternate days. It always occurred after feedings and was projectile in type. Symptoms increased in severity and frequency up to the time of admission despite all possible variations in formulae and antispasmodics. Refeeding was instituted successfully, the patient never having vomited after this procedure. Shortly before admission peristaltic waves were noted in the epigastrium, passing from left to right. The weight had increased 970 Gm. An interesting point in the family history is that one year before the birth of the patient, the mother had had an exploratory laparotomy, at which time congenital bands causing a partial intestinal obstruction were released.

Physical examination was essentially negative. The only abnormalities noted were areas of capillary hemangiomata over the face and trunk and a somewhat protuberant

abdomen showing peristaltic waves in the epigastrium, coursing from left to right. There was some anemia; red blood cells, 3,160,000; hemoglobin, 11.0 Gm.

Operation was performed September 21, 1935, under ether anesthesia. The stomach and pylorus were normal. The duodenum was dilated two to three times its normal size and was tightly bound down by a series of peritoneal adhesions. These were freed, allowing the duodenum to assume its normal position. The abdomen was closed with silk.

The patient tolerated the operative procedure well. She was given a transfusion and frequent subcutaneous saline infusions following operation. Feedings were given by gavage, the tube being left in place due to the presence of some distension, which soon subsided. There were two normal stools on the second postoperative day. The patient was discharged on the third postoperative day. Her subsequent course has been entirely satisfactory and without symptoms.

Case 7.—No. 97611. N. B., a white male infant, age two days, was admitted to the Strong Memorial Hospital December 21, 1934, for repair of cleft palate and hare lip. Physical examination was negative except for the local condition, *i.e.*, complete left unilateral hare lip and a complete bilateral cleft palate.

The patient vomited every feeding following admission. Stools consisted mostly of meconium with a small amount of formed stool. Changes in formulae and antispasmodics were without avail. Five per cent glucose was given subcutaneously several times each day. Four days after admission, on December 25, a small amount of barium was given. Fluoroscopy showed marked gastric retention with only a minute amount of barium passing through the pylorus. Most of the barium was still present in the stomach after three hours. There was no dilatation of the duodenum. Conservative treatment was continued without any improvement until December 28, when operation was performed.

Operation was started under local infiltration, but it was necessary to change to drop ether in order to adequately explore the abdominal cavity. The stomach, pylorus, and duodenum were normal. The colon was reflected upward and Treitz's ligament identified. Just beyond Treitz's ligament was a marked twisting of the upper jejunum due to hyperperitonealized bands. Salt solution injected into the stomach flowed readily through the pylorus and the duodenum but stopped at Treitz's ligament. The peritoneal bands were divided, releasing the intestinal kinking. There still seemed to be an obstruction at one point. A membrane obstructing the lumen was suspected and the bowel was opened longitudinally. No intrinsic obstruction was found and the opening was closed by suturing transversely. The injected saline then flowed readily beyond this point.

The patient continued to vomit for three days following operation during which time numerous glucose and saline infusions were given. Beginning on the fourth day, feedings were well taken and retained except for an occasional attack of vomiting. Feeding of course was difficult due to the cleft palate. However, the patient's condition improved steadily so that by January 15 the cleft premaxilla was approximated by silver wire under ether anesthesia. The patient expired suddenly at 5 P.M. the day of operation.

Postmortem examination, besides a large frothy bloody mucous mass filling the posterior pharynx, showed focal areas of bronchopneumonia, acute bronchitis, subpleural ecchymoses, and hemangiomata of the tricuspid and mitral valves.

Case 8.—No. 83494. B. C., white, female, age ten months, was first admitted to the Strong Memorial Hospital, December 20, 1933, at the age of four months because of persistent vomiting since birth. The vomiting occurred as often as five to six times per day, three to four hours after feeding, and was not projectile. The vomitus consisted of curdled, partially digested milk, and did not contain bile. The amount varied considerably but for the most part represented only a small amount of the previous feeding taken. Stools had been normal. Physical examination was entirely negative. The blood and urine were normal. Fluoroscopy of the intestinal tract after barium showed a large atonic stomach with marked delay in emptying. She was readmitted

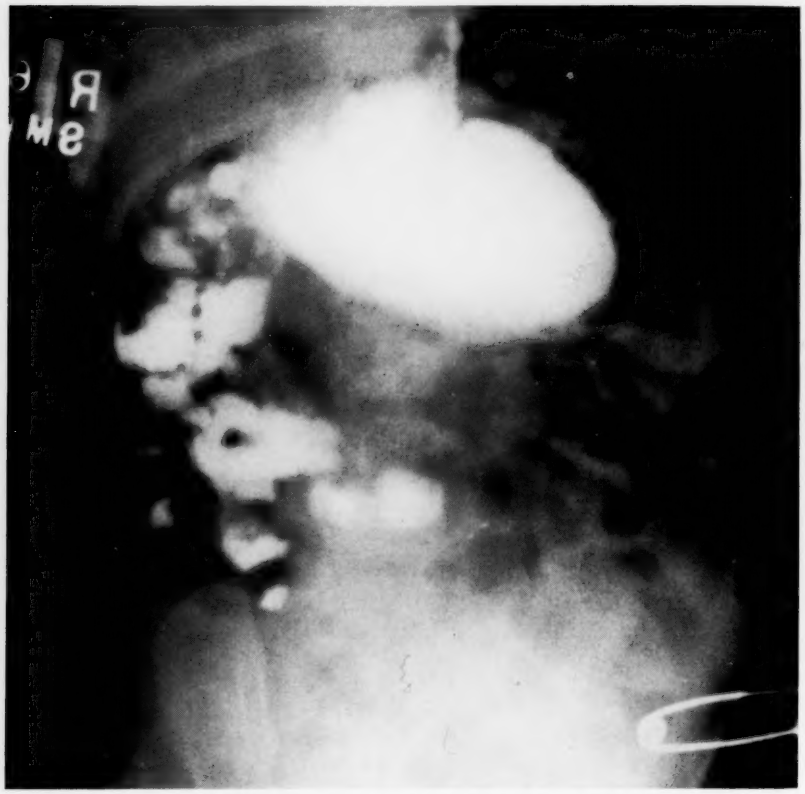

FIG. 5.—Case 8. Roentgenogram showing passage of barium into small intestine, but delay in an egg-shaped loop seen through the gas filled stomach.

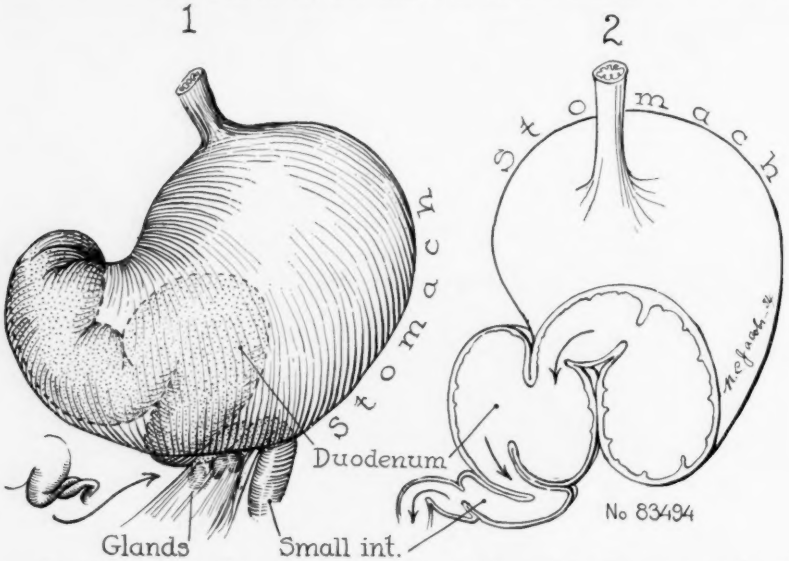

FIG. 5a.—Case 8. The condition found at operation explains the queer egg-shaped shadow in the roentgenogram. This is a torsion and plication obstruction freed by releasing the bands.

for the same symptoms on June 11, 1934, having been carried along on a medical regimen during this time. Vomiting was less frequent. On one occasion peristaltic waves were seen. However, for a week or two prior to admission vomiting increased and the patient lost one pound in one week. In the interim, symptoms had changed somewhat. Vomiting frequently occurred immediately after feeding, and at times was projectile in type. Bile had been present in the vomitus on two occasions. There had been increasing constipation.

Physical examination showed no abnormalities. Abdominal examination was negative. The blood and urine were negative. Examination of the gastro-intestinal tract after the injection of barium showed no dilatation of the stomach but considerable delay in emptying, a fairly large amount being present after six hours (Fig. 5).

Operation was performed June 13, 1934, under ether anesthesia, a right rectus incision being made. The stomach and first portion of the duodenum were dilated. The transverse and ascending colon were freed from the lateral abdominal wall and rotated to the left, giving a good exposure of the retroperitoneal duodenum. In the third portion of the duodenum behind the mesentery there was a longitudinal torsion of the intestine in two places, caused by hyperfixation. The duodenal attachments were freed in these places allowing the duodenum to straighten out. Gas promptly passed into the first portion of the jejunum. The abdomen was accordingly closed (Fig. 5).

The postoperative course was satisfactory though somewhat difficult. Subcutaneous saline infusions were given as indicated. Feeding was resumed the evening of the day of operation. Vomiting occurred at irregular intervals, but never was persistent or alarming. The patient was discharged from the hospital 15 days after operation in excellent condition.

The patient was last seen October 23, 1935, one year and four months after operation. Her condition was excellent and development had been normal. The only vomiting spell noted since the first two weeks after discharge was associated with an acute upper respiratory infection.

Case 9.—No. 89644. R. S., a baby boy, was born in the Strong Memorial Hospital May 21, 1934. He appeared normal. The birth weight was 6.9 lbs. In the first few days of life he vomited mucus on several occasions. Upon nursing at the breast he regurgitated small amounts after each feeding. On the fifth day he vomited once forcefully. The stools were entirely meconium for the first three days and there were no stools for the next two days. The weight dropped till on the fifth day it was 5.4 lbs. He became jaundiced and dehydrated. General examination was negative. The physicians thought that he might have pyloric stenosis and dehydration jaundice. He was given repeated saline infusions. Several different formulae were tried. Atropine 1:1000 was used, one drop before each feeding. In spite of this the vomiting continued and on one occasion it contained a small amount of blood. He was given one teaspoonful of barium in two ounces of his formula and observed under the fluoroscope. The mixture seemed to be delayed at the cardia for some time and was then regurgitated. An esophagoscope was accordingly passed through the cardia and no obstruction was encountered. The pediatricians then placed the child on a thickened formula, each feeding being preceded by gastric lavage. The vomiting seemed to become less in amount for a time. This period of apparent improvement was followed by recurrence of symptoms in a more severe form. It was necessary to give daily infusions. An abscess developed, at the site of one of these infusions, in the left axilla. A second barium study did not give information of value concerning the upper gastro-intestinal tract but small flecks of barium given seven days previously were observed in the rectum, indicating that obstruction was not complete. The child passed small stools containing bile daily. He did not gain in weight but remained at about 6.6 lbs., being supported by daily infusions. No peristaltic waves were observed at any time and no tumor was made out by palpation. A diagnosis of partial obstruction in the duodenum either due to atresia or faulty attachment was suggested by the surgeons. Exploration was carried out June

19, 1934, under drop ether anesthesia. The stomach and the first portion of the duodenum were distended. The pylorus was normal. The right transverse colon was freed from its peritoneal attachments from the midline to the hepatic flexure. Further mobilization was made down to the cecum and the whole right colon was then retracted toward the left, allowing exposure of the root of the mesentery. There were a series of angulations in the retroperitoneal duodenum where peritoneal attachments produced an accordion pleated effect (Fig. 6). These bands were released and the duodenum traced until it emerged into jejunum under the mesenteric vessels. There were no signs of atresia. Closure was made in layers with silk.

Convalescence was complicated by continuation of vomiting in small amounts, but a gradual improvement was effected by careful regulation of his formula. He was discharged in good condition September 17, 1934, at which time he weighed 9.7 lbs. He was in the hospital in November, 1934, for otitis media. His weight at this time was 11.1 lbs. A bilateral myringotomy was done with relief. On February 28, 1935, he was admitted to the hospital because of a conjunctivitis and an acute purulent left otitis media. He was taking his food without trouble. His weight was recorded as 15.1 lbs. The baby was examined again on April 22, 1936. He was eating and sleeping well. He was regarded as normal. His weight was 22.6 lbs. at this time.

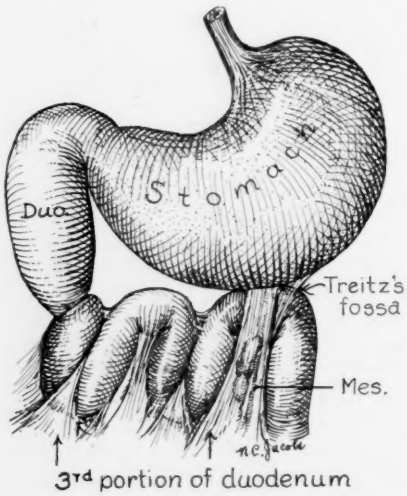

FIG. 6.—Condition found at operation in Case 9. This is an "accordion-pleated" obstruction due to hyperfixation.

Case 10.—No. 79531. M. M., a white female infant, third child, seven weeks old, prematurely born, was admitted to the Strong Memorial Hospital September 8, 1933, because of persistent vomiting since birth. Three hours after birth the baby "threw up red stuff" according to the mother. The character of the vomitus remained unchanged for four days, following which it consisted only of feedings previously taken. The symptoms persisted despite all possible changes in formulae and antispasmodics. The vomiting occurred immediately after each feeding and had consisted of a substantial portion of the feeding. Vomiting was definitely projectile. Constipation was marked, the infant having but one small yellow formed stool per day. The weight on admission was the same as the birth weight, 2,600 Gm.

Physical examination was entirely negative except for undernourishment and a palpable liver and spleen, each of which was 1 cm. below the costal margin. Laboratory examination showed some anemia; red blood cells, 3,700,000; hemoglobin, 80 per cent. The urine was negative. The Wassermann was negative.

All possible conservative measures were tried. The vomiting persisted, and even increased in frequency and amount. On one occasion four days after admission peristaltic waves were seen in the epigastrium passing from left to right. This was noted a second time on September 18. Gastro-intestinal fluoroscopy showed retention, no barium leaving the stomach during an hour and a half of observation. By this time the child's general condition appeared worse than on admission and laparotomy was performed for relief of pyloric obstruction.

Operation.—September 21, under ether anesthesia. A small right rectus incision was made. Because the stomach was dilated, the pyloric muscle was divided although no true tumor was present. Although an adequate explanation of the vomiting was not

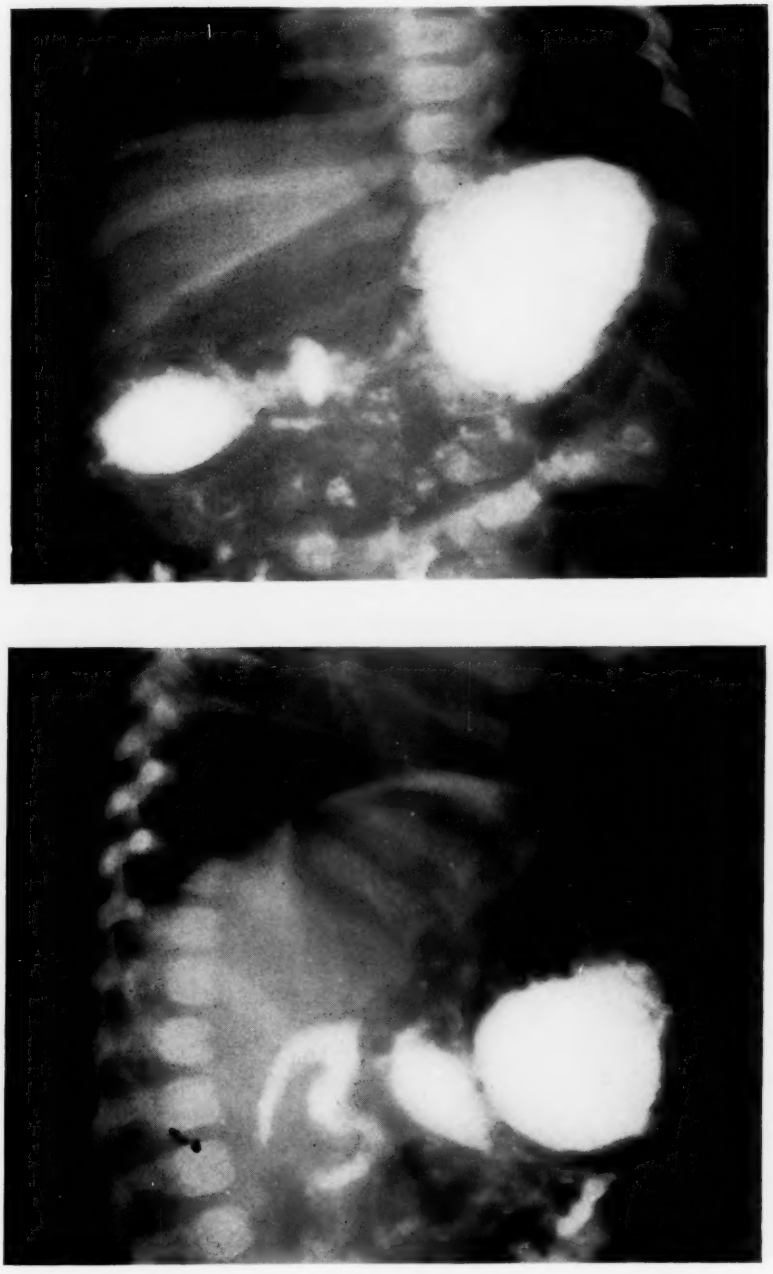

FIG. 7.—Case 11. Anteroposterior and lateral roentgenograms. Showing dilated stomach with folding of antrum and duodenal cap into posterior position. There is a twist of the normal duodenal curve.

demonstrated, the operation was terminated at this point because of the extremely poor condition of the patient.

Following operation the patient was given a transfusion and subcutaneous saline. During the first week after operation the patient's condition improved and vomiting was only occasional. However, beginning on the eighth day vomiting again became regular and frequent and on the following day coffee ground material, which gave a 3+ benzidine reaction, was vomited. A second transfusion was given. The patient vomited blood a second time. Her general condition was poor. The weight on October 3 was 2,550 Gm.

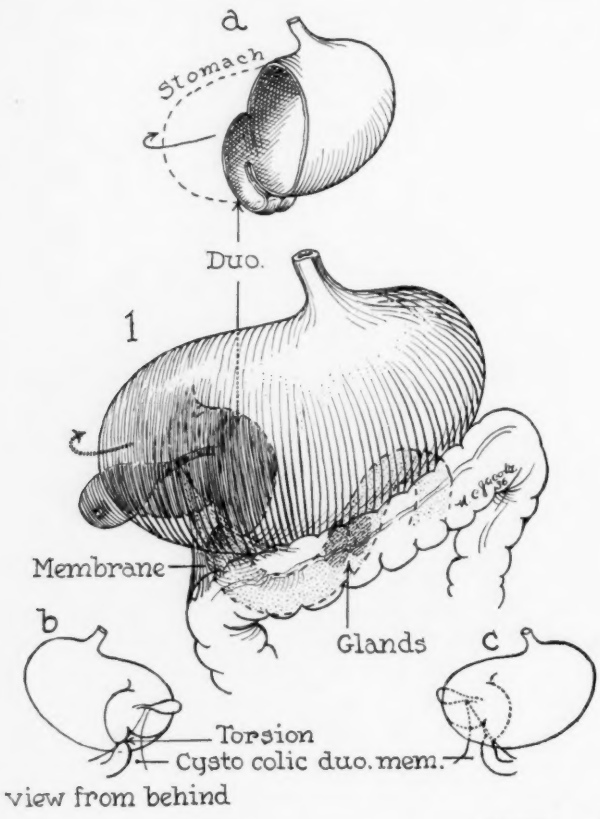

FIG. 7a.—Case 11. The condition found at operation explains the roentogenographic appearances. The obstruction in this case was due to a band running from the duodenum to the third part of the duodenum and colon.

A second laparotomy was performed October 4 under ether anesthesia, the previous incision being opened and considerably enlarged. The stomach was dilated. The cecum and appendix were in close approximation to the second portion of the duodenum to which they were attached by peritoneal bands which caused a kinking and obstruction of the duodenum. These bands were divided, releasing the cecum, which was placed in its normal position. A systematic exploration of the entire small bowel showed other peritoneal bands. About four inches from the ileocecal valve other peritoneal bands were found. These involved the ileum and transverse colon in a complicated knot which was released by division of the bands. About six inches proximal a similar condition was found. At Treitz's ligament there was found a triple twist of the bowel,

causing a loop to kink upon itself with an acute angulation when it entered a retroperitoneal position before emerging into the first portion of the jejunum. This portion of the intestine was intimately associated with the blood supply and consequently it was difficult to straighten it out. After correcting this last abnormality, it was noted that there was a three inch opening in the mesentery of the transverse colon, a congenital defect, as the edges were smooth and regular and contained the blood supply. This rent was carefully closed, following which the intestines were returned to the abdominal cavity and the wound closed.

The patient tolerated the operation extremely well considering her previous poor condition and the unavoidable shock associated with the manipulation. She responded well to an immediate transfusion. There continued to be vomiting but in general it was less frequent and regular and not projectile. The amount vomited was small. Her convalescence was retarded by numerous recurrent upper respiratory infections. In December bilateral otitis media was responsible for a temporary set back. Despite the occasional vomiting, however, the patient showed a general tendency to improve. Her weight increased slowly but steadily, on a moderately thickened formula, and at the time of discharge January 12, 1934, she weighed 4,300 Gm., an increase of 1,700 Gm. over her weight on admission. She was last seen a month after discharge, at which time she weighed 4,660 Gm. During the month she had vomited three or four times.

Case 11.—No. 110306. W. B., a baby girl, was admitted to the Strong Memorial Hospital December 26, 1935, because of vomiting. She was two weeks old at this time. The mother had had an uncomplicated pregnancy and no difficulties at delivery. The child's birth weight was 9 lbs. She was the tenth child in the family. Three of the children before this one had had vomiting, controlled by thickened feedings; one sister had died in infancy from vomiting and diarrhea of two months' duration after many different types of feeding had been tried without success. The present child was fed on the breast for only a few days. A breast pump was then used and the milk fed by bottle. On the tenth day a dilute whole milk formula was substituted for the mother's deficient milk supply. Vomiting was infrequent during the first two weeks. Every feeding was followed in 15 or 20 minutes by a moderate sized well formed stool. Five days before admission the baby became constipated. Stools were obtained only by glycerine suppositories. This child had lost one pound in weight. Examination showed nothing remarkable. She was given a modified formula and sent home. Within the next week she had diarrhea and a marked mastitis was present. She had projectile vomiting on January 7, 1936, and was readmitted January 8.

A suggestion of peristaltic waves in the epigastrium was made out by one observer and confirmed by several others during the next few days. She was placed on thickened feedings. Atropine was tried also but had to be discontinued. The baby continued to vomit and had diarrhea for several days. The symptoms then let up and she gained slightly in weight. She was discharged January 20, 1936. After being home for only a few days, there was a recurrence of symptoms. She regurgitated small amounts after each feeding; had six or more slimy movements a day; cried constantly when awake; and acted very hungry. The mother was reluctant to have her in the hospital but was worn out by the care of the baby during the next ten days. Accordingly the child was readmitted January 31, 1936. Peristaltic waves were easily made out after she had taken a few swallows of water. In a few days all her vomiting and diarrhea had been controlled. Arrangements were made to continue the treatment at home under supervision of a public health nurse. She was discharged February 6. She retained most of her food during the next ten days. And gained weight up to 10.2 lbs. But ten days later she had lost a little because of vomiting, with alternating constipation and diarrhea. A gastro-intestinal barium study was made March 2. She was three months old at this time and had not gained an ounce in the last month. The stomach was greatly distended and atonic. It folded back on itself over the pyloric region so that the antrum and pylorus were pointing toward the left and posteriorly. The duodenum occupied a peculiar twisted position. The

obstruction was partial since only half the barium remained in the stomach after four hours. The diagnosis of faulty fixation of the duodenum on the mesentery with partial obstruction to the stomach was made (Fig. 7).

At operation under drop ether anesthesia March 9 a greatly dilated redundant stomach was found. By gentle traction on the stomach the antrum and pylorus were exposed. The pyloric ring was in spasm which gave the impression of a hypertrophic stenosis, but it soon relaxed. The first portion of the duodenum was slightly larger than normal. On exposing the mesentery by freeing the transverse colon, it was evident that the large bowel had not migrated completely to the right side but was attached just beyond the midline in the region of the gallbladder. The gallbladder was large, and a well defined cystocolic membrane ran across it to the hepatic flexure region of the colon causing a torsion of the descending portion of the duodenum which was caught in this membrane. On freeing it the duodenum straightened out (Fig. 7*a*). The duodenum was traced all the way to its emergence from under Treitz's ligament without finding any other anomaly. Closure was made in layers with silk. Postoperative course was satisfactory. There was no vomiting. Feedings were resumed rapidly and the patient was discharged March 23, 1936. She weighed 11 lbs. at this time. She has had no symptoms since operation. She takes five to six ounces five times a day and is still not quite satisfied. Her weight at 4½ months, on April 16, 1936, was 12.4 lbs.

Symptoms and Signs.—The most constant symptom presented by every one of these patients is vomiting. It may have been noted a few hours after birth. Usually, however, it is observed only after the first feedings. The vomiting is usually fairly regular. It may occur immediately after a feeding or not till some time within three to four hours. In most cases it begins mildly but progresses to a more violent projectile type as time goes on. Vomiting may be irregular with free intervals of apparent improvement, or it may become steadily more frequent. The vomitus may consist simply in the ingested material, water, milk curds, mucus, or it may contain "coffee ground" material, or blood as noted in Cases 1, 2, and 11. In a doubtful case the presence or absence of bile in the vomitus has been used as evidence of an obstruction above or below the bile papilla. We have not found this examination very valuable; in fact, in this series, chemical or microscopic tests of the vomitus have scarcely ever been recorded.

The presence of blood or "coffee ground material" in the vomitus is considered pathognomonic of duodenal atresia or stenosis. On the other hand, the presence of bile in the vomitus is not a reliable diagnostic sign in localizing the obstruction as it has been demonstrated that enough bile can find its way through the microscopic openings to be readily detectable. Also, as Cordes pointed out, accessory bile ducts may be present entering the intestine above and below the obstruction. The detection of bile in the meconium is likewise subject to misinterpretation for the same reasons and also because of the fact that, while bile secretion begins normally about the third month of gestation, these congenital obstructions may not develop until much later. Consequently, despite a complete obstruction below the ampulla of Vater, the first bowel movements after birth may and often do contain bile which has reached the lower intestine before the establishment of the obstruction.

While normal meconium may be obtained, complete obstipation and absence of milk stools is the rule. If bile is present in appreciable amounts in the vomitus it aids in the differentiation, being absent in pyloric stenosis.

As a sequel to the vomiting there may result an upset in the bowel elimination. The usual trouble is constipation with a tendency towards small dry stools. Occasionally diarrhea has occurred; and alternating periods of constipation and diarrhea. In a number of cases the stool has been normal. Some of these obstructions are only partial in nature so that no absolute rule can be formulated. Blood was recorded in the stool examination in only one instance.

When the vomiting has been persistent, the baby either loses weight rapidly, or he fails to gain under the best of supportive treatment. The weight loss in some instances has been as high as one-quarter of the original birth weight in ten days to two weeks. But it is remarkable that these newborn babies can be carried along for a considerable period, just as adults can be in high obstructions, provided that adequate water and salt are supplied. In some cases where the vomiting has not occurred after every feeding the babies have even shown some slight gain in weight. This is usually a testimonial to the care and skill of the attending pediatrician.

Upon examination the signs of dehydration are frequently observed. These babies appear puny and cyanotic when severely ill. The infants may also show other developmental abnormalities (Cases 1, 6, and 7), or may be prematurely born (Cases 5 and 10). Peristaltic waves passing from left to right have been seen at some stage in examination of these infants in a majority of instances. In Case 1 a tumor was visible occupying a transverse position. When vomiting has been severe there may be signs of alkalosis, manifested by tetany and convulsions.

Course.—The pediatricians attempt to regulate the feedings of these babies by varying their formulae; substituting different materials; changing the frequency or consistency of the feedings; feeding by tube; expressing air from the gastro-intestinal tract and refeeding; and the use of antispasmodics. In some instances the babies are found to be habitual air swallowers and regurgitators and must be broken of the habit. Rarely, there also seems to be a protein sensitiveness to milk in some infants. Fluids and salts can be supplied parenterally as necessary when the vomiting is not controllable by other means. Transfusions may be of value. We cannot praise too highly the skill and resourcefulness of our pediatric staff in the management of these infants. When, in spite of all efforts, vomiting continues, an attempt to localize the lesion accurately by roentgenologic examination seems warranted.

Roentgenologic Examination.—Although it is admitted that a barium mixture should not be used above an obstruction, we have made use of a thin mixture of barium in most of these cases. The information which it gives us in this group seems to outweigh its disadvantages as we have decided to operate usually in any case because of the persistence of symptoms. The

barium can be evacuated by lavage or by vomiting when operation has to be delayed for any reason. In Cases 1, 2, and 4 characteristic dilated atonic stomach and duodenum shadows indicated that the obstruction was near the third portion of the duodenum (Figs. 1, 2, and 4). In Case 3 a peculiar sausage shaped shadow along the lesser curvature attracted our attention. This child was thought to have hypertrophic pyloric stenosis until the roentgenogram helped us visualize the true lesion. The parallel shadows of this herniated loop can be seen through the gastric shadow on this one film of the series (Fig. 3). Roentgenograms in Cases 5, 7, and 9 showed delay in gastric emptying but gave no other help in the diagnosis. In Case 8 the dilated duodenum showed retention and a clear rounded outline after the barium had left the stomach. It clearly indicated that the partial obstruction was in the duodenum behind the stomach (Fig. 5). In Case 11, by use of the lateral view, it was possible to recognize that the greatly dilated stomach had folded back upon the pyloric antrum; and that there was a peculiar angulation in the duodenal loop leading away from this region (Fig. 7). We think that this information together with the fact that it sometimes shows that obstruction is not complete is of value to us. We have never yet seen any deleterious effects from the use of this method.

Differential Diagnosis.—The only real problem in diagnosis is to differentiate these cases from those of congenital hypertrophic pyloric stenoses. Congenital hypertrophic pyloric stenosis usually occurs in males and usually in the first born child in the family. The onset of vomiting is rarely at birth. It usually appears some time during the second to the sixth week. In a majority of cases the vomiting increases in severity, becoming projectile in character. Peristaltic waves passing from left to right can be made out in about three-quarters of the cases. A palpable tumor can be felt with varying success, this depending to some extent upon the importance attached to it by the examiner. The other symptoms and signs—loss of weight, dehydration, alkalosis, constipation and scanty urine are common to the two conditions. A study of congenital hypertrophic pyloric stenosis in our clinic showed that it occurred in males in 90 per cent; that it had been noted in the second child in the family once; in the third child twice; and once in the sixth child of the family; that vomiting started at birth in one instance; and that it did not start till two months of age in another; the rest of the cases following the rule; that peristaltic waves were observed in 80 per cent; and that a palpable pyloric tumor was made out in just half of the cases. In our cases of congenital obstruction there were seven females to four males. The majority also were not the first born in the family. There was one first born girl and three other possible first born babies though this point was not stated in the history. The others ranged from the third to the tenth child in the family. The vomiting came on early—usually before the first week, sometimes during the first day, and often following the first ingestion of anything. Two of the children were prematurely born; and three showed other congenital abnormalities. Peristaltic waves were made out in

seven of the 11 cases but no tumor was palpated in any of them. The roentgenogram had diagnostic value in six of the 11 cases. It would appear from this review that we should look with suspicion upon any baby which starts vomiting during the first week of life, especially if of the female sex and with other signs of congenital anomalies. If no relief can be obtained by medical measures we should be prepared to give a thin barium mixture for roentgenologic appraisal of the cause. The presence of peristaltic waves with the absence of a palpable tumor might be regarded as of some significance in helping to make the diagnosis. The presence of bile or blood in the vomitus would be in favor of some lesion other than hypertrophic pyloric stenosis.

Intussusception can be ruled out by its usual onset still later in life than that of hypertrophic stenosis; and by the typical acute picture it presents.

Strangulated inguinal hernia must sometimes be differentiated but in our experience this has not been difficult.

Prognosis.—Intestinal obstruction in the new born due to congenital abnormalities is not uncommon. The fact that a large percentage of such cases reported in medical literature is based on postmortem examination is evidence of the extremely high mortality associated with this condition. A universal appreciation of the comparative frequency of its occurrence will tend to improve results as the unfavorable outcome in many instances is directly attributable to incorrect diagnoses or to diagnoses established too late for surgical intervention to be successful.

In a certain percentage of cases of intestinal atresia (15 per cent according to Davis and Poynter), the congenital defects are multiple and beyond the aid of surgery. Furthermore, many of the defects occur in the duodenum and upper jejunum (194 of the 392 cases in the series of Davis and Poynter), thus placing them in the category of high intestinal obstructions. This, superimposed upon the fact that the new-born infant is not a good operative risk, mitigates against improvement in results in such cases. Admitting a necessarily high mortality upon the basis of these facts, there still remains a definite percentage of fatal results which can, conceivably, be eliminated by early correct diagnosis and the prompt institution of surgical treatment. An organic mechanical obstruction demands early operation for its relief. Since many of these congenital defects are amenable to operative treatment and this fact can be established only at the time of operation, all cases so diagnosed should be given the opportunity of relief which only operation affords.

The prognosis in cases of congenital occlusion of the small intestine is extremely poor. The first successfully treated case is credited to Ernst[14] who operated upon an infant ten days old for congenital duodenal atresia, relieving the condition by a "duodeno-entero-anterior anastomosis." Eleven months postoperatively the patient was progressing normally. In 349 published articles there are reports of 29 cured cases: 15, in which the obstruction was due to malrotation, and 14, in which the obstruction was intrinsic,

atresia or stenosis.[5] In the 60 cases reported by Ladd the mortality was 70 per cent. The mortality in cases of malrotation was 55 per cent (nine cured of a total of 20 cases) and in cases of intrinsic obstruction it was 80 per cent (eight cured of a total of 40 cases). These results showed a great improvement when one considers the statement of Thorndike[13] who placed the mortality of congenital duodenal atresia at 99 per cent. In the present group of 11 cases there has been only one death, a mortality of 9 per cent.

The prognosis in cases of congenital anomalies about the mesentery depends to a large extent upon the alertness of the attending physician. These babies can stand a considerable amount of fluid and salt loss if it is compensated for by parenteral medication. It is also true that in some cases the obstructions are only partial so that the baby gets some nourishment. When the child loses weight or fails to gain because of the amounts lost by vomiting, steps should be taken to correct the underlying fault if possible. The surgeon also must bear his share in getting these babies by the operation. This requires gentle handling of tissues more than any other single thing. The fate of these babies depends then upon the preoperative condition—a reflection of the care given by the attending physician; the operative manipulation and judgment—a reflection on the care of the attending surgeon; and the continued combined efforts of both physician and surgeon in the postoperative follow up. In our series the results have been excellent. All the babies have been carried through the operation to relieve their obstructions without a fatality. There have been two deaths, one almost two years after the operation, from pneumonia; the other as the result of a second operation for cleft palate two weeks after the duodenal obstruction had been relieved satisfactorily. This baby was a great feeding problem which forced action against better judgment. The postmortem examination showed bronchopneumonia which may well have been due to aspirated food.

Treatment.—Preliminary preparation for operation is exceedingly important. Loss of weight and dehydration must be combated by restoring the water balance. This may be done by the intravenous, intraperitoneal or subcutaneous routes. Blood, water, glucose and salt may be used as circumstances require. We usually fluoroscope these infants to see whether an enlarged thymus is present. If so, operation would be delayed for a few days following roentgen therapy. Usually no preoperative drugs are necessary. Occasionally atropine in 1/400 to 1/200 gr. doses is given. We have used local infiltration in the past but when there has been crying, straining and evisceration of the bowels, so much damage has been done in restoring the viscera to the peritoneal cavity that we have abandoned this practice. Drop ether carefully given by a well trained anesthetist, we believe to be the anesthetic of choice. A very small quantity will often suffice. Most writers agree that babies do not stand hemorrhage, shock or infection well.[19, 20, 21] For these reasons during operation there must be most careful hemostasis; gentleness in handling tissues; lack of exposure to prevent heat loss; and progession without delay so that a minimum of anesthesia is necessary.

Emphasis should not be placed on speed in these operations. Babies will stand operations well for one and one-half hours or longer if necessary. However, the more time that a surgeon can save, without sacrificing gentleness, the better. Respect for the tissues is by far the most important factor in these operations. The flashy, speedy, rough surgeon will not do well with these patients. Abdominal closure is made in layers with fine silk. We have had no disruption of these wounds and no infection. The dressing is made light and sealed in with waterproof adhesive.

In congenital absence of a segment of duodenum, obviously the relief of the obstruction by operation is the only treatment possible. A short circuiting operation, duodenojejunostomy or gastro-enterostomy, is the logical procedure. The choice between these two procedures will vary with the operator and the conditions encountered in the individual case. In those cases in which there is a diaphragm stretched across the lumen of the bowel,

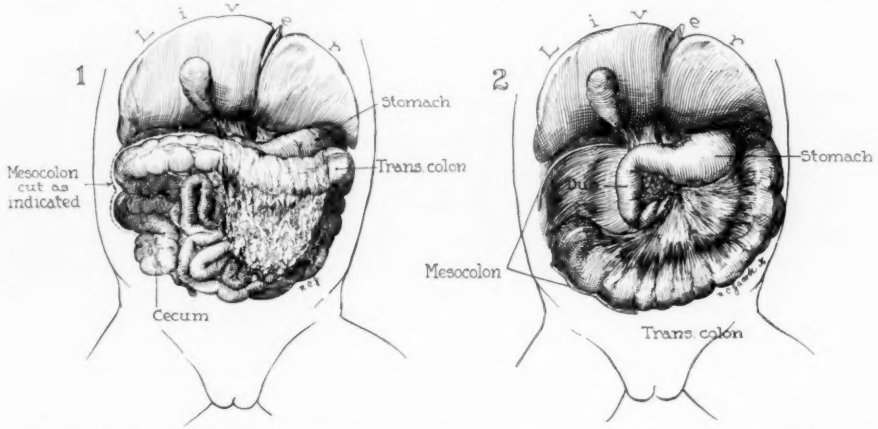

FIG. 8 (1 and 2).—Represents the operative approach to the root of the mesentery. The transverse and ascending colons are freed from their lateral attatchments and the retroperitoneal duodenum exposed. The condition illustrated in 2 was present to some degree in Cases 5, 6, 7, and 10.

longitudinal incision of the intestine, removing the diaphragm and transverse suturing, as proposed by Morton[15] and performed by Petersen,[16] should be seriously considered as being a simpler procedure than intestinal anastomosis. However, it is often difficult to definitely establish the presence of such a diaphragm. Under no circumstances should an enterostomy (jejunostomy) be performed, as it is only a palliative procedure which is not well tolerated by infants. In those cases in which it has been done a rapid death resulted. Clogg[17] has made a valuable suggestion that, in as much as congenital intestinal defects are prone to be multiple, one should inject fluid into the collapsed bowel below the obstruction, forcing it along to discover the presence of any additional defects. This possibility should always be kept in mind, as it might easily condemn to failure what otherwise would be an entirely successful operation.

The operation of choice for the extrinsic anomalies due to faults in migration, descent, and fixation, is not to attempt a gastro-enterostomy or

entero-enterostomy as has been done so often in the past. The surgeon should realize that this is the type of obstruction which can be successfully untangled if he knows how to get at it. The best way to unravel these puzzling anomalies is to get a clear view of the mesentery. This can be done only by detaching the transverse and ascending colons from the parietal wall and rotating them toward the midline, which gives excellent access to the root of the mesentery (Fig. 8). This operation we had worked out independently in our clinic only to find that Ladd had been using the identical procedure. We believe that this operation will result in a decided improvement in the operative mortality in these extrinsic anomalies. It stands in much the same relationship as the division of the pyloric sphincter did to cases of pyloric stenosis with the consequent abandonment of the previous posterior gastro-enterostomy operations.

The surgeon should not enter the abdominal cavity with a preconceived idea that he is going to undertake a certain procedure. It is most difficult to abandon the plan to fit circumstances which may not demand it. It is especially difficult not to wish to simply divide the pyloric muscle in some of these cases.

After operation special nursing care is of great importance. The baby must be kept warm, but not so hot that he will lose his fluids through perspiration. Cold draughts must be avoided. Fluids should be restored again. Codein may be used for pain. Distention should be combated with stupes, rectal tubes and later enemata. Babies do not stand pain and distention well. Their reactions to these are violent and alarming. Lavage may also be necessary. Acidosis may be prevented by the use of glucose. The baby should be restored to his normal environment as rapidly as his condition warrants. His dressings must be kept dry, especially during the first week. Measures should be taken to seal off the urinary tract from the operative site by suitable apparatus. It is a pleasure to acknowledge the close cooperation of Dr. Samuel Clausen and his staff in the after care of these patients.

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SPLENECTOMY FOR HEMOCLASTIC CRISES*

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DURING the clinical course of certain blood diseases, namely, congenital hemolytic icterus and thrombopenic purpura, there occur episodes of increased blood destruction. These are known clinically as "crises." They may develop spontaneously or be precipitated by various environmental factors. In congenital hemolytic icterus the increased destruction affects chiefly the erythrocytes. In thrombopenic purpura there may be an increased destruction of thrombocytes. Although splenectomy is ordinarily the treatment of choice in the more chronic stages of these hemoclastic dyscrasias, it has been thought,[7, 9, 11, 12, 13] and it is the accepted surgical teaching[4, 5, 6, 8, 10] at the present time, that the acute exacerbations are a direct contra-indication to splenectomy.

Previous studies within our group[1, 13, 14] have provided evidence which incriminates the spleen as one of the principal factors in the physiologic control as well as in the pathologic destruction of the circulating blood elements. If such be the case, then, during exacerbations of splenic activity, even to the extent of "crisis" proportions, removal of the spleen is not only desirable, but in many instances may become essential to survival. Nevertheless, the risk involved in undertaking any major abdominal operation with a total red count rapidly approaching the one million mark, has, heretofore, interdicted splenectomy under such circumstances.

In addition to the conviction that the major rôle was played by the spleen in the diseases under discussion, our early studies revealed, as a part of the postsplenectomy mechanism, an immediate marked postoperative rise in both the red blood cells and platelets, which amounted essentially to an autotransfusion. The specificity and significance of this response have been discussed[1] and form the basis for our having undertaken a series of splenectomies during active hemoclastic crises without as yet encountering one operative fatality.

It is the purpose of this paper to present the pertinent clinical evidence from eight patients each successfully splenectomized during an acute hemoclastic crisis. Six of the patients had congenital hemolytic icterus; two had thrombopenic purpura.

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CASE REPORTS

Congenital Hemolytic Icterus

Case 1.—H. S., a mail carrier, age 50, was admitted to the Research Service of the University Hospital, December 4, 1933, presenting splenomegaly, severe epigastric pain and deep jaundice.

As a boy he had noted his sallow skin. He was advised also of an anemia, and was aware that he fatigued easily. About 20 years ago he was diagnosed as having "splenic anemia." He had already noted the mass in his upper left quadrant. He was hospitalized, about 19 years ago, for what appears to have been a subacute exacerbation. Splenectomy was advised at that time but was not accepted. Since then, he has presented the characteristic picture of chronic congenital hemolytic icterus. During the past three years the disease has gradually become more severe, and in addition, mild symptoms of cholelithiasis have appeared.

On November 29, 1933, while delivering mail, he experienced acute, severe epigastric pain, which disabled him. Jaundice became evident the next day, and grew progressively deeper. His food intake was decreased and an unusual weight loss ensued. An associated acute hemoclastic crisis was suspected, and he was referred to the Research Service by Dr. Paul A. Adams.

His past history reveals mainly the story of congenital hemolytic jaundice with the characteristic ebb and flow of its minor symptomatology. During the past few years his appetite has diminished and an associated weight loss has ensued. To his knowledge none of his family except his son has had evident icterus. This son, an icteric, frail boy of 15, with marked splenomegaly, was subsequently (July 12, 1934) successfully splenectomized on the Research Service for this same disease.

Physical examination of the patient revealed marked weakness, enlarged spleen, anemia and a deep jaundice. T., 98.2°; P., 66; R., 15; B. P., 140/70. The sclerae were deep yellow. The spleen extended 4 cm. below the umbilicus. The liver was enlarged and tender. There was definite right epigastric tenderness. The urine was negative, other than for the high bile content. The stool was not acholic and was negative for blood. Roentgenologic examination revealed a small annular shadow, about 1 cm. in diameter to the right of the upper body of the third lumbar vertebra. The gallbladder was not visualized.

The significant blood findings are presented in Chart 1. There was no significant anemia (red blood cells, 3,820,000; the reticulocytes were 4 to 6 per cent); a microcytosis (mean red blood cell diameter 6.9, mean red blood cell volume 100 cu. microns), and an increased fragility of the red blood cells (0.582 to 0.300) were found. The icterus index was 882, the highest we have ever recorded. The patient was Type B blood group. On December 5, the direct van den Bergh was immediately positive, and the indirect was 21 mg. During the 48 hours subsequent to admission the red count fell to 3,000,000 and the reticulocytes gradually increased to 14.2 per cent. The icterus index decreased appreciably, to 428 (Chart 1).

The findings thus indicated an icterus of double origin; an older, congenital hemolytic form with splenomegaly, and a more recent acute, acquired form due to obstruction by a common duct stone. Since about two-thirds of patients with hemolytic icterus develop pigment calculi within the gallbladder, such an occurrence is readily understandable and should be anticipated. There was evidence of only moderate hemolysis. The preoperative icterus index was 440, predominantly of obstructive origin.

Two courses of management were considered: (1) Splenectomy, to stop an impending hemoclastic crisis, and (2) a preliminary cholecystostomy, in an attempt to relieve the acute and severe cholemia. The latter seemed the more threatening. It was clearly realized, however, that the cholecystostomy might provoke even more severe hemolytic activity by the greatly enlarged spleen.

The relatively good level of the red cells and hemoglobin (Chart 1), together with

the evidence of but moderate hemolysis, influenced us to follow the second course in the hope that the obstructive jaundice might subside somewhat before the onset of a more acute hemolysis. The patient was prepared for cholecystostomy by the administration of 5 cc. of 10 per cent calcium chloride in 200 cc. of normal saline intravenously, and by a hypodermoclysis of 1,500 cc. of Ringer's solution containing 5 per cent glucose. This was given the evening before, and repeated the morning of the laparotomy.

Cholecystostomy was accomplished uneventfully on December 7, under local anesthesia, using a nitrous oxide-oxygen supplement. The abdomen contained considerable bile-stained ascitic fluid. The liver was greatly enlarged. It was greenish, finely nodular and the lobules were unusually prominent. The gallbladder was enlarged, and its walls were thickened and injected. It compressed readily, and contained numerous stones. The cystic duct was about 7 Mm. in diameter when flattened. It appeared to be patent. The common duct was tense and distended. At its lower portion, well above the ampulla, was a hard, irregular mass about 15 x 10 x 10 Mm. This was thought to be a stone, although it was not explored with a needle. The spleen was huge and moderately adherent. The splenic capsule was thick, white and opaque. The adhesions were soft and vascular.

Opening the gallbladder fundus revealed the thick, fibrous wall, a rather normal yellow-brown bile and numerous characteristic pigment stones, which varied up to 18 Mm. in diameter. They were black, somewhat elastic, friable and most irregular in shape. They were removed and the cystic duct explored. It was patent. The gallbladder was drained. Careful hemostasis was effected, and there was but a moderate loss of blood. The patient was returned to his room in good condition with the blood pressure 78/44.

Progressive relief from the cholemia ensued. The icterus index, 24 hours postoperative, fell to 120 (Chart 1). On the other hand, hemolysis definitely increased. On the evening of December 9, 36 hours postoperative, the red count had fallen to 1,610,000 and the hemoglobin to 3.9 Gm. The reticulocytes had risen to 20 per cent. An acute, even fatal, hemoclastic crisis threatened.

From our previous experience it seemed evident that splenectomy was indicated as being the most reasonable procedure to arrest the unusually severe, although expected complication. We could also, with reasonable assurance, depend upon the customary postoperative autotransfusion as a restorative measure. An emergency splenectomy was performed, therefore, December 9. The patient was prepared for this as for the cholecystostomy. Local anesthesia, supplemented by nitrous oxide-oxygen, was employed. Adrenalin, 1 cc., was administered intramuscularly about 20 minutes before clamping the splenic pedicle. This was to ensure maximum contraction of the smooth muscle in the splenic capsule and trabeculae.

The spleen was removed without incident through a left subcostal Kocher incision. During the procedure the blood pressure, increased by adrenalin, remained elevated around 140/50. There was no evidence of circulatory failure and prompt postanesthetic recovery ensued.

The spleen weighed 1,740 Gm., and measured 22 x 15 x 9 cm. It was firm, and apparently contracted. The capsule was in places as thick as 4 Mm. and an opaque white. The splenic pulp was increased. It was dark red, cellular, friable and more fibrous than normal. Microscopically the splenic corpuscles were infrequent, and the pulp spaces were filled with blood. The phagocytic clasmatocytes were increased.

Immediately following operation the characteristic and expected autotransfusion ensued, and the reticulocytes decreased (Chart 1). The icterus index continued to decrease, and reached 30 on December 14. Convalescence was then uneventful until December 13, when the patient became disoriented, arose and walked about the room. A chill followed, and later a fever and leukocytosis (Chart 1). Vomiting supervened. There was no bile in the vomitus. *B. coli* infection of the splenectomy wound was apparent December 15. It was drained and treated by dakinization. The skin flaps separated and on December 22 a secondary closure was undertaken under local anesthesia.

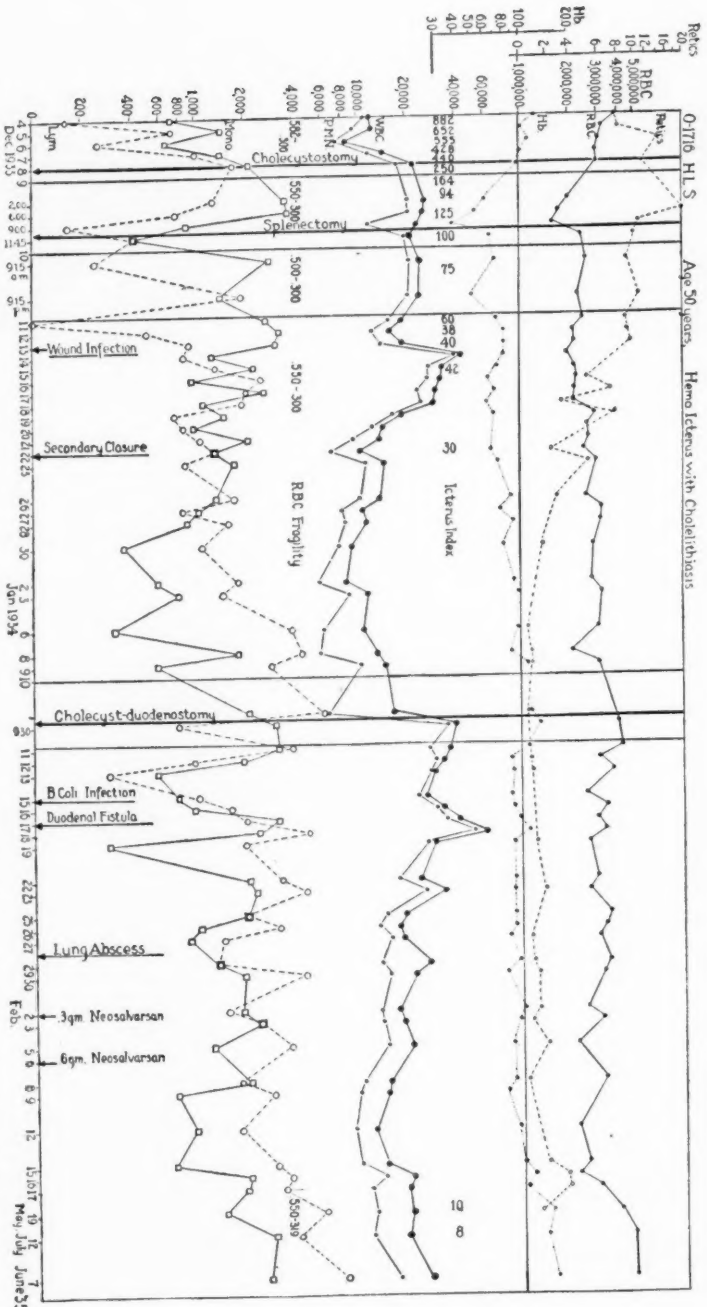

CHART 1.—(Case 1) Splenectomy for the hemoclastic (erythroclastic) crisis of congenital hemolytic jaundice. (Journal A.M.A.)

Dakinization was continued and sound healing, without subsequent herniation, eventually occurred.

Biliary drainage continued; however, there was no evidence of autodigestion about the cholecystostomy opening. The original rubber tube was later replaced by a mushroom catheter which minimized the leakage. Convalescence was steady and afebrile.

On December 27 the biliary tract was visualized by injecting lipiodol through a small catheter inserted into the cholecystostomy opening. The gallbladder, also the cystic, hepatic and common ducts were examined fluoroscopically. The lipiodol came to an abrupt stop, 6 cm. down the dilated common duct. Roentgenograms revealed an irregular block in the region of the shadow which was inferred to represent the common duct stone. There was a questionable leak past the stone into the intestine. A subcutaneous tract was also demonstrated connecting the cholecystostomy margin with the infected splenectomy wound. This was possibly the original avenue of infection. No reaction followed this investigation.

The bile draining from the cholecystostomy showed no evidences of containing pancreatic secretion, which apparently entered the duodenum normally as shown by aspiration through a duodenal tube. Thus an irregular, occluding common duct stone remained, definitely above the ampulla of Vater and which did not obstruct the external pancreatic secretion. The increased risk of removing this stone, by mobilizing the duodenum medially, performing a choledochotomy, and of possibly inserting a T tube, was weighed. To be sure, the cholecystostomy assured temporary biliary drainage. Eventually, however, owing to the continued fluid and mineral loss, it required closure. Cholecystoduodenostomy was finally elected as the best solution of the problem. This was accomplished January 10 under local anesthesia, using supplementary nitrous oxide-oxygen.

The cholecystostomy opening was dissected free, the gallbladder fundus mobilized, and the stoma trimmed. The gallbladder fundus was then attached to the adjacent duodenum and a two row cholecystoduodenostomy accomplished. The suture line was then fastened to the fascia of the gallbladder bed with interrupted silk sutures. A Penrose drain was left just under the skin. Ephedrin was given at the end of the operation. The blood pressure was 88/66. The pulse was fair, rate 112. There were no evidences of shock.

During the ensuing two days the temperature rose to 104.8°; pulse, to 160; and increased respirations. These all gradually subsided. On January 14 the patient expectorated a foul smelling, thick, tenacious mucus. On January 15 a *B. coli* infection of the right abdominal wound was noted. This broke down and on January 17 a duodenal fistula became established. Bile and duodenal juice were identified. The skin edges became eroded. The duodenal fistula was treated by plugging, strapping, care of the skin edges and the use of a light cradle. It soon began to fill in and was closed by February 6.

On January 19, cough and considerable foul smelling sputum production were noted. This continued. A roentgenologic examination January 26 revealed a left hilus shadow suspicious of lung abscess. This was confirmed by a stereoscopic examination January 31. A fluid level was evident. The abscess wall was thin. Adequate postural drainage was instituted. Because of the character of the sputum on February 2 and again on February 6, neoarsphenamine was administered. The sputum production began to decrease February 4. It was only 10 cc. in amount by February 10, and ceased on February 12. Stereoscopic examination, February 16, showed no evidences of any residual lung abscess, and a clear parenchyma. General improvement ensued. The patient's appetite returned, and he gained weight. The patient was discharged February 17.

At no time during this stormy convalescence, subsequent to the splenectomy, was the hemolytopoietic equilibrium seriously disturbed. The bone marrow met all demands promptly and efficiently as they arose (Chart 1). There were no evidences of decompensation or incapacity. This is of significance in evaluating the fundamental nature of congenital hemolytic icterus.

Since leaving the hospital the patient has gained 40 pounds (18 Kg.). Both

operative wounds are well healed without herniation. There have been thus far no evident gastro-intestinal symptoms referable to the results of the extensive abdominal surgery. The patient resumed his occupation as mail carrier, June 1, 1934. July 12, 1934, the day splenectomy was successfully accomplished on his son, the father's red blood cells were 4,850,000, hemoglobin 14.2 Gm., color index 0.95, reticulocytes 1.2 per cent, icterus index 8, hematocrit 52, erythrocyte fragility 0.550-0.319 and leukocytes 10,000, with an essentially normal differential percentage. After two and one-half years the patient continues to remain clinically well.

Case 2.—B. Y., a housewife, aged 56, was admitted to the University Hospital February 9, 1935, presenting great weakness, an unusual pallor, moderate jaundice and an enlarged spleen.

Since childhood she had noted that she very easily became fatigued, that even minor ailments disabled her greatly, and that she had a peculiar pallor. At one time she was thought to have pernicious anemia. Four months ago she developed an upper respiratory infection. This resulted in an increase in the anemia and the development of recognizable jaundice. During the succeeding interval there have been remissions and relapses with, however, a progressive increase in weakness and pallor. One son of 28 has had manifest jaundice during the past year. There is evidence of generalized arteriosclerosis. A cerebral vascular accident two years ago resulted in a residual hemifacial paralysis with ptosis of the eyelid and some atrophy of the right side of the tongue.

Physical examination revealed a striking exhaustion. She was poorly oriented. The loose, pale skin indicated considerable weight loss. Pitting edema was noted over the tibiae and sacrum. The retinal arteries were tortuous and striped. Hearing was diminished, particularly on the right. The bony thorax was somewhat immobile with a decreased vital capacity and other evidence of pulmonary emphysema. Crepitant râles were constantly heard over the posterior right base. The heart revealed an occasional premature ventricular systole. The pulmonary second sound was accentuated. A short, soft blowing systolic murmur was audible over the base. T., 98.0°; P., 100; R., 15; B. P., 110/60. The palpable arteries were moderately sclerosed. The enlarged and firm spleen extended to just below the umbilicus.

Neurologic examination by Dr. Harry LeFever revealed a right facial weakness (supranuclear); weakness of the left masseter and pterygoid muscles; paresis of the right upper eyelid; deviation of the tongue to the right; impaired hearing on the right with bone conduction greater than air conduction; and unequal reflexes. These findings were interpreted as the result of a previous brain stem lesion, probably a hemorrhage.

Laboratory investigation confirmed the characteristic findings of congenital hemolytic icterus; an anemia, red blood cells 1,480,000, hemoglobin 5.0 Gm.; a reticulocytosis of 54 per cent; microcytosis; and increased fragility of the red blood cells, .319–471. The icterus index was 60. There was a leukopenia, 3,300 to 3,800. The blood N.P.N., glucose, and carbon dioxide combining capacity were normal. The urine revealed no bile but pathologic amounts of urobilin; it was otherwise normal. The stools contained bile pigment but occult blood was not demonstrable. The Wassermann and Kahn tests were negative. The direct van den Bergh test was negative; the indirect was 4.2 mg.

Upon this evidence a diagnosis of congenital hemolytic icterus, without biliary obstruction, seemed warranted. On the other hand, destruction of the red blood cells was gradually becoming more marked. During the first four hospital days the erythrocyte count decreased from 1,620,000 to 1,310,000, while the icterus index rose from 60 to 75; and the reticulocyte count from 54 to 70 per cent. These findings signified increasing hemolysis and indicated the onset of a "crisis," and an unfavorable course of the disease. The blood typing showed Group O; however, from our previous studies transfusion seemed contra-indicated. Since at this time three emergency splenectomies for hemoclastic crises had already been successfully accomplished, we had less hesitancy in advising this operative procedure as a means of arresting the threatening hemolytic process.

Kidney function tests were within normal limits. An E.K.G. revealed a normal

sinus rhythm with an occasional sino-auricular premature contraction; and left ventricular preponderance. Roentgenologic examination of the chest revealed increased bronchovesicular markings. A flat film of the abdomen revealed evidence of gallstones.

Splenectomy was accomplished uneventfully February 13. Preoperatively morphine gr. ¼ and atropine gr. 1/150 were administered. The usual left rectus incision was made after local novocaine infiltration anesthesia; and nitrous oxide-oxygen was administered during the exploration and splenectomy. Considerable yellow tinged ascitic fluid was encountered. The distended, thick-walled, opaque, and moderately adherent gallbladder contained three large calculi. Palpation of the biliary ducts revealed no evidence of stones. A small accessory spleen 15 Mm. in diameter was removed from the posterior splenic pedicle.

The spleen weighed 811 Gm. and measured 23 × 16 × 6.5 cm. Supravital studies of the pulp revealed the usual increased phagocytosis of the red blood cells by clasmatocytes, atrophy of the lymphatic elements, and engorgement of the splenic parenchyma by erythrocytes. Grossly and microscopically the pathologic appearance was that of congenital hemolytic icterus. The usual postoperative autotransfusion was immediately apparent. Within the first 24 hours the erythrocytes had increased to 2,030,000 and the hemoglobin to 5.8 Gm.; with 11,900 leukocytes. Other than for some productive cough, subsequent convalescence was steady and uneventful. The icterus diminished and gradually disappeared. The reticulocyte count decreased. The patient's strength slowly returned and she was discharged March 6, 1935, 21 days postoperative, greatly improved.

Case 3.—O. L., a white housewife, age 52, was admitted to the Research Service June 7, 1935, presenting a purulent otitis media complicated by a progressively increasing anemia and a recently developed icterus.

A little over a year previously, while under hospital observation for goiter, a marked leukopenia and a moderate, sustained anemia and thrombopenia had been discovered. The white blood cells ranged from 1,200 to 2,500; the erythrocytes from two to three million. During the past year various medical regimens had been instituted in an attempt to overcome this status, with little apparent result. It was believed that her bone marrow was moderately hypoplastic. About two weeks ago, however, she developed an acute purulent otitis media, which ruptured the right tympanum, with a resultant profuse foul smelling discharge. Two days before admission a right facial palsy supervened. With the establishment of this infection more evident anemia developed, accompanied by definite clinical icterus for the first time. The leukopenia became more pronounced.

Upon arrival at the hospital the total leukocyte count was only 200. Hemolytic streptococci were cultured from the middle ear discharge. Direct smears showed contaminating bacilli and cocci. The icterus index was 40. The direct van den Bergh was negative; the indirect showed a trace. The red blood cell count was 1,200,000, with 18 per cent reticulocytes. The platelets were greatly diminished.

Owing to the atypical picture which she had presented during the past year, suggesting a hypoplastic bone marrow, a transfusion of 400 cc. of citrated blood was given, cautiously, soon after admission. This was followed by a chill and a severe reaction.

Upon physical examination a splenomegaly, hitherto absent, was discovered. The fragility test revealed some decrease in the resistance of the erythrocytes to hypotonic solutions. The icterus index had increased to 60. With evidence now available of an increasing hemolytic anemia, of increased erythrocyte fragility, of a rising reticulocyte count, of splenomegaly, of a mounting icterus index and of an even more marked leukopenia—the conclusion that we were dealing with an atypical congenital hemolytic icterus seemed warranted. Further investigation strengthened the view that we were now facing a definite hemoclastic crisis initiated by the middle ear infection, and splenectomy was determined upon.

On June 8, 1935, a 920 Gm. spleen was removed without incident under local anesthesia with supplementary nitrous oxide-oxygen. The liver and gallbladder appeared to

be normal, as did the other viscera. No accessory spleens were seen. Upon supravital study the splenic pulp was found to be greatly engorged with neutrophilic leukocytes, in sharp contradistinction to the extreme peripheral leukopenia. The phagocytic clasmatocytes were greatly increased in number.

Convalescence was complicated by the development of a large rectal abscess with surrounding necrosis from which hemolytic streptococci, similar to those recovered from the otitis media, were cultured. This was treated by hot moist packs, by incision and drainage; and the danger of general sepsis was combated by repeated daily small blood transfusions (from June 11 to June 16). The rectal area healed slowly.

On July 3, 1935, the erythrocyte count was 3,610,000 with only 1.4 per cent reticulocytes. The leukocyte count, however, remained low—1,800—for some time. The patient was discharged July 6, 1935, definitely improved. She has been followed during the past year. On July 8, 1936, just one year after leaving the hospital, her blood picture showed: red blood cells, 4,250,000; hemoglobin, 13.0 Gm.; reticulocytes, 0.2 per cent; leukocytes, 5,100. This restoration to normal of the previously depressed blood findings is further evidence that we were dealing with the splenomegaly of congenital hemolytic icterus.

Case 4.—L. H., a white housewife, age 31, was referred to the Research Service by Dr. C. C. Fitzpatrick of Jackson, Ohio. She was admitted January 1, 1936, critically ill; and presented the picture of an acute hemoclastic crisis, *in extremis*.

Her early history is that of unrecognized congenital hemolytic icterus. She was in apparent good health up to about four years ago, when she had a cesarean section to terminate her third pregnancy. At that time an anemia and a splenomegaly were noted. Liver therapy was tried without appreciable effect. She was twice hospitalized and transfused. About a month ago liver therapy was resumed and six injections of liver extract were given. This resulted in some questionable improvement. One week ago, however, she was confined to bed because of increasing anorexia and weakness. Two days ago persistent nausea and vomiting developed. The resultant extreme dehydration and demineralization complicated the clinical picture on admission to University Hospital, with evidence of recent rapid loss of weight, of increasing anemia, with moderate jaundice and progressive dyspnea.

Physical examination revealed a critically ill patient. The skin was pale and icteric; the mucosae and finger tips were cyanotic; there was moderate dyspnea with orthopnea; no petechiae or other evidences of bleeding could be found, yet the anemia was profound. The sclerae were yellow. T., 99.6°; P., 122; R., 32; B.P., 118/50. The pulse was full. A blowing systolic murmur was audible over the entire precordium. The liver was moderately enlarged. The spleen extended to the iliac crest, and was tender. A pitting edema was demonstrable over the tibial crests. There were no abnormal neurologic findings.

Immediate examination of the blood revealed red blood cells, 800,000; hemoglobin, 2.1 Gm.; reticulocytes, 16 per cent; leukocytes, 10,800; platelets greatly decreased, and an icterus index of 60. The urine showed an albuminuria. The N.P.N. was 40 mg. per cent; the blood glucose 160 mg. per cent; the plasma CO_2 30 vol. per cent; and the blood chlorides 205.

Intranasal oxygen was instituted. This resulted in some improvement of the cyanosis and dyspnea. A small indirect transfusion was given about three hours after admission. This was accompanied by chilliness, temperature elevation and some reaction. Later sodium bicarbonate and glucose were given intravenously and after four hours 500 cc. of citrated blood. Only slight transitory improvement was noted.

The relative lack of response in this critically ill patient, during the first 12 hours of medical treatment, left no alternative but splenectomy. While the rationale of this operative procedure had been established in our previous experiences, no patient had presented heretofore quite so critical a condition. It was decided to explore and limit the procedure to ligature of the splenic artery, in case adhesions or operative difficulties made the actual removal of the spleen too hazardous.

On January 2, 1936, without any preoperative medication, 150 cc. of novocaine containing eight drops of adrenalin per 100 cc. was infiltrated along the left upper rectus region. The spleen, exposed through an adequate left rectus incision, was large, bluish and fortunately nonadherent. Nitrous oxide-oxygen was then administered. The spleen was luxated and its pedicle freed. One-half cubic centimeter of adrenalin was next injected directly into the splenic artery. A definite, visible contraction of the spleen followed, thus squeezing blood cells from the large splenic reservoir into the circulation and effecting an autotransfusion. The pedicle was then clamped and tied and the spleen removed. Several accessory spleens, ranging from 3 Mm. to 3 cm. in diameter, were then removed from the region of the pedicle.

Immediate improvement occurred in the patient's clinical status, which allowed the abdomen to be thoroughly explored. The liver was enlarged. Its lobules were evident; however, it did not appear to be cirrhotic. The gallbladder was enlarged, firm, thickened and contained numerous irregular stones, which are so characteristic of the calculi occurring in instances of congenital hemolytic icterus. The ducts appeared to be patent. The stomach and colon were normal, and the pelvis was negative. The patient left the operating room definitely improved.

The spleen weighed 1,635 Gm. and measured 26 x 15 x 10 cm. Grossly it was characteristic of hemolytic icterus. Microscopic sections revealed a diffuse fibrosis and numerous dilated endothelial channels. Increased phagocytic activity was evident, as well as increased blood pigment. The splenic corpuscles were atrophic.

Postoperatively the blood pressure remained over 100 systolic. Hematology: red blood cells, 1,710,000; white blood cells, 23,700; hemoglobin, 3.9 Gm.; platelets, 350,000. Hypodermoclysis and intranasal oxygen were administered. At the commencement of the second day 600 cc. of citrated blood were given to hasten the clinical recovery. From that time on convalescence was progressive and uneventful.

On February 22, three weeks postoperative, the blood findings paralleled the clinical improvement. Red blood cells, 4,490,000; hemoglobin, 13.4 Gm.; white blood cells, 7,200; reticulocytes, 0.4 per cent; and platelets 1,890,000. The icterus index was also normal. The patient was discharged January 28. Six months later, on July 22, she was clinically well. Red blood cells, 5,420,000; hemoglobin, 14.9 Gm.; reticulocytes, 4 per cent; leukocytes, 8,400; icterus index, 6.2; and thrombocytes, 1,900,000.

Case 5.—G. P., female, age 4, with an acute severe hemoclastic crisis, was splenectomized September 6, 1933, by Dr. V. A. Dodd.[1]

Case 6.—V. T., female, age 20, with an acute hemoclastic crisis, was splenectomized March 25, 1935, by Dr. Luke Zartman.[1]

Thrombopenic Purpura

Case 7.—E. M., a colored schoolgirl, age 17, was admitted to the University Hospital June 23, 1934, for the management of purpura with an attendant severe anemia. Unusual pallor together with scattered subcutaneous and submucous hemorrhages were evident.

She had been well and active up to early May, when unusual bleeding from the gums was first noted. This became more marked. Menstruation early in June was prolonged and profuse. An increasing pallor, with the symptoms of progressive anemia, caused her to consult a physician. She was hospitalized elsewhere June 14, and the presence of a severe anemia confirmed. A blood transfusion was given June 16. This resulted in but temporary relief, since the metrorrhagia continued and in addition epistaxis and hematemesis developed.

Her past history revealed a long recognized tendency to easy bruising. Nevertheless, she had undergone tonsillectomy eight years previously without any complications. No other incidence of unusual bleeding was known to have occurred in her family.

Upon entering the hospital the patient was dyspneic, somewhat cyanotic and perspiring profusely. T., 103°; P., 120; R., 48; and B. P. 103/?. Her pallor was striking.

Cutaneous petechial hemorrhages up to 5 Mm. in diameter were evident. The eyes reacted normally and the sclerae were clear. There were no retinal hemorrhages. The gums were spongy and hemorrhagic. Ulceration had developed about a carious and abscessed lower right molar, and had extended into the adjacent tongue and soft palate. Necrosis with resultant fetor oris was evident. Recent bleeding was also noted in the nasal cavities. The heart was negative, other than for a blowing systolic murmur heard at the apex. Pelvic examination revealed a cervical polyp and uterine bleeding. Other examinations were negative. The spleen was not palpable, and apparently not enlarged.

Investigation of the blood (Chart 2), revealed 1,770,000 red blood cells, hemoglobin 4.9 Gm. (Newcomer), color index 0.9, reticulocytes 57 per cent; erythroblasts 1,200; and normoblasts 2,400 per cmm.; mean erythrocyte diameter 8.6 microns with both anisocytosis and poikilocytosis extreme on direct examination but with no "sickling," immediate or delayed; erythrocyte fragility was markedly decreased, hemolysis started at 0.319 and was not complete until something less than 0.210 salt equivalent; icterus index 6; leukocytes 30,000, with 67 per cent actively motile, mature, neutrophilic granulocytes, 5 per cent myelocytes C and metamyelocytes, 1 per cent eosinophils, 18 per cent lymphocytes, and 9 per cent monocytes; blood platelets 3,500; bleeding time 23 minutes; coagulation time (Howell) 11 minutes; calcium time 12 minutes; prothrombin time 12 minutes; no clot retraction in 24 hours; tourniquet test (50 Mm. of mercury) showed innumerable petechiae after ten minutes. The Wassermann and Kahn tests were negative; carbon dioxide combining power 36; blood Group O. Doubtless due to the oral infection and anemia the daily temperature ranged from 99° to 104° F. Blood cultures were negative.

The striking thrombopenia together with the coagulation findings pointed to the diagnosis of idiopathic thrombopenic purpura hemorrhagica. Elsewhere a diagnosis of aplastic anemia with symptomatic purpura had been entertained. However, both the red and white cells in the circulating blood revealed such evident regenerative responses that an aplastic bone marrow was at once ruled out. The unusually high reticulocyte count (57 per cent) even resembled that seen occasionally in congenital hemolytic icterus, or in pernicious anemia after liver therapy. A sternal bone marrow puncture yielded no added information. The nucleated red cell response (3,600 per cmm.) added further evidence of unusually active erythropoiesis. Even after an epinephrine test June 26 (Chart 2), blood platelets did not appear in the peripheral circulation. In fact, platelets were essentially absent throughout the entire period of preoperative observation (Chart 2).

Intensive medical treatment was first instituted. During the first 24 hours 50 cc. of whole blood were given intramuscularly. On the second day 500 cc. were given intravenously. Viosterol, a high fat-protein diet, large amounts of orange juice daily, oral hygiene, and even cauterization and packing of the cervical canal were of little avail, since the hemorrhagic tendency and anemia steadily increased. Splenectomy was finally advised, but was refused until the fourth hospital day when the red blood cells following a profuse hemorrhage fell to 725,000 and the hemoglobin to 2.4 Gm. The nucleated red cells had increased to 6,000. The leukocytes were 25,000, the reticulocytes 30 per cent and *no platelets were to be found in the peripheral blood.* An emergency transfusion of 700 cc. of citrated blood was given to the comatose and moribund patient, which increased the red blood count to 1,230,000. A second of 400 cc. was given later in the day. The following day (June 28) the general clinical condition of the patient was greatly improved. Red blood cells, 1,660,000; hemoglobin, 4.9 Gm.; leukocytes, 10,800; platelets, 0; T., 99.6°; P., 88; R., 24; and B. P., 100/58. It was thereupon decided to resort to surgery without further delay and to attempt ligation of the splenic artery, if splenectomy seemed inadvisable.

On June 28 splenectomy was accomplished uneventfully with a minimal loss of blood. A left rectus incision was made under local anesthesia, which was supplemented by nitrous oxide-oxygen, for the intra-abdominal manipulation. No free fluid was

encountered in the abdomen. Petechial hemorrhages, however, were numerous and were increased by the necessary handling of the viscera. The spleen was normal in size, appearance and position. Its pedicle was short. It weighed 225 Gm. At the conclusion of the operation the pulse was 96, respirations were 24, the blood pressure was 140/90; red blood cells 1,990,000, reticulocytes 15 per cent, hemoglobin 5.9 Gm., leukocytes 14,800, and blood platelets 21,900. Twenty-four hours after the splenectomy the leukocyte count was 52,000 with 85 per cent active mature neutrophils, red blood cells 2,230,000, hemoglobin 6.2 Gm., and blood platelets 89,000 (Chart 2). All bleeding had ceased.

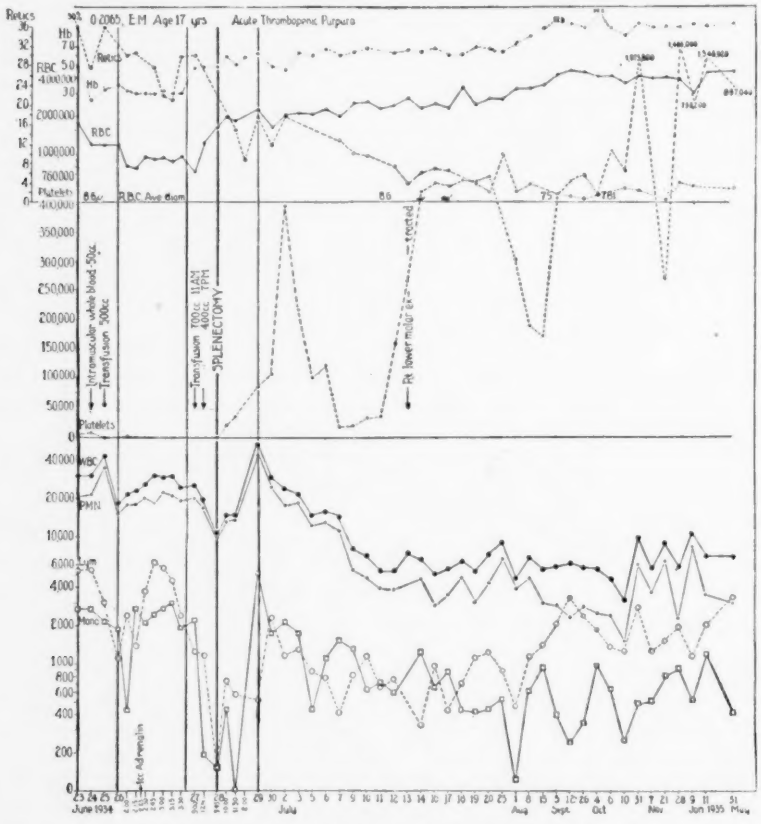

CHART 2.—(Case 7) Splenectomy for the hemoclastic (thrombocytoclastic) crisis of thrombopenic purpura. (Journal A.M.A.[1])

Investigation of the fresh splenic pulp by the supravital technic failed to reveal free extracellular clumps of blood platelets. The débris in the clasmatocytes presumably represented whatever of these elements had reached the spleen. This permitted an explanation for the failure of platelet increase subsequent to the preoperative injection of epinephrine. Microscopically sections of the spleen revealed a suggestion of chronic inflammation. The splenic corpuscles were prominent but normal. The clasmatocytes did not appear to be increased in number or unusually phagocytic. The reticulum was hypoplastic; the endothelial cells and the vascular bed were not remarkable. The splenic pulp was not engorged with red blood cells as occurs in hemolytic icterus, nor with blood platelets, as is described in certain purpuric states.

Immediately following the splenectomy the blood platelets increased in number until on the fourth day postoperative they attained a count of 395,000. During this period

they varied greatly in individual size. During the succeeding eight days a gradual decrease ensued without notable change in the patient's condition. By the twenty-second postoperative day they had reached 748,000 (Chart 2).

With the cessation of the hemorrhages and the attainment of satisfactory convalescence the ulceration of the mouth cleared rapidly. The abscessed molar was extracted on the fifteenth postoperative day and the gums were then given needed attention. Both the hemoglobin and red cells gradually increased. At the end of three weeks there were 3,500,000 erythrocytes. The reticulocytes promptly decreased (Chart 2). The bleeding time became normal, clot retraction occurred promptly and the tourniquet test (ten minutes at 50 Mm. mercury pressure) failed to cause petechiae. By the ninth day postoperative the elevated temperature and pulse had returned to normal. The patient was discharged July 20, on the twenty-second postoperative day, in good condition.

During the past two years she has maintained an entirely normal existence, with a platelet count sustained consistently at about one million per cmm. (Chart 2). She

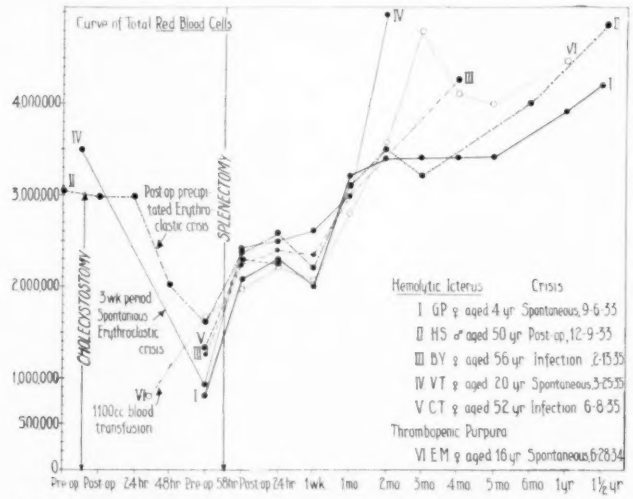
Curve of Total Red Blood Cells
4,000,000
3,000,000
2,000,000
1,000,000
500,000
0
CHOLECYSTOSTOMY
SPLENECTOMY
Post op precipitated Erythroclastic crisis
3 wk period Spontanious Erythroclastic crisis
1100cc blood transfusion
Hemolytic Icterus Crisis
I GP ♀ aged 4 yr Spontaneous, 9-6-33
II HS ♂ aged 50 yr Post-op, 12-9-33
III BY ♀ aged 56 yr Infection 2-15-35
IV VT ♀ aged 20 yr Spontaneous, 3-25-35
V CT ♀ aged 52 yr Infection 6-8-35
Thrombopenic Purpura
VI E M ♀ aged 16 yr Spontaneous, 6-28-34
Pre op Post op 24 hr 48 hr Pre op 5 8 hr Post op 24 hr 1 wk 1 mo 2 mo 3 mo 4 mo 5 mo 6 mo 1 yr 1½ yr

CHART 3.—The results of splenectomy for hemoclastic crises in six cases. (Journal A.M.A.)

has married and, on March 21, 1936, gave birth to a normal female child. Throughout this stay under observation in the University Hospital, from March 20 to 30, 1936, no evidence of recurrence of the purpuric state was noted. The platelet count remained high. There was no unusual bleeding either previous to or after labor. The puerperium was quite normal. She continues to remain clinically well.

Case 8.—A. J., a housewife, age 61, with an acute hemoclastic crisis occurring in the course of thrombopenic purpura, was successfully splenectomized June 30, 1936. She was dismissed in good condition on July 30. This splenectomy was accomplished after the completion of this paper.

CONCLUSIONS

The evidence presented by these eight patients, all successfully surviving a major surgical procedure during a hemoclastic crisis, further supports the rationale of splenectomy in the treatment of properly selected and prepared patients suffering from hemolytic icterus or thrombopenic purpura of splenic origin. There have, thus far, been no failures. We attribute this success to careful differential diagnostic studies, optimum preoperative management

and the application of modern surgical knowledge to the problem. The implications of this study add materially to our knowledge of the pathologic physiology of the spleen.

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AN ANATOMIC RESTUDY OF THE PELVIC LYMPHATICS*

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THE two principal problems of the proctologist are infection and malignancy. In each of these processes the lymphatics are important factors. In a subsequent paper the possible relationship between lymphopathia venereum and rectal stricture will be presented; in that paper use will be made of the findings set forth in the present report.

An excellent account of the pelvic lymphatic structures could be crystallized from the various studies that have been made. However, because of the lack of any particular interest on the part of the authors in clinical proctology, no one of the well known studies correlates this field. Several of the modern French workers are interested in lymphatics as they pertain to the spread of malignant disease, and in so far as this interest pertains to the lower bowel, their studies are very informative. Of the considerable study that was made of the literature, only cursory mention can be made, later in the paper.

The present anatomic restudy of the genito-anorectal lymphatics was begun in order to correlate the more general studies with the special point of view of the proctologist. In order properly to evaluate the field it has been necessary to acquire first hand familiarity with actual anatomic preparations. A series of these has been prepared.

Anatomic Terminology.—Dr. Collier F. Martin has repeatedly stated that much confusion exists with regard to the terminology of anorectal anatomy. The appended nomenclature is that favored by Doctors Martin and Batson and is significant anatomically, physiologically, pathologically and clinically:

The anus is the external opening of the anal canal (a rima in the anatomic sense).

The anal canal represents the terminal portion of the large intestine, from the anus to the anorectal line, a distance of approximately 2.5 cm. The entire lining is modified squamous epithelium.

The term "anal skin" is used to replace the misnomer "anal mucous membrane."

The terms "anorectal line," "pectinate line," "dentate line," "dentate margin," and "mucocutaneous junction" are synonymous. They all refer to the boundary line between the anal canal and the rectum, comprising the anal papillae with the intervening delicate margins of the anal valves.

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The terms "white line of Hilton" and "intersphincteric line" are synonymous, but they were thought by Hilton, and by many others, to be synonymous also with "anorectal line." Hilton's white line is more readily felt than seen. It is definitely within the boundaries of the anal canal; it lies approximately half way between the anus and the anorectal line. In fact, in many individuals, the intersphincteric line is rendered distinct only during digital examination of the anus, the finger displacing the internal sphincter upward, so that temporarily the internal and external sphincter muscles become actually superior and inferior muscles, respectively. This displacement during examination probably accounts for the errors which are to be seen in some drawings.

The material studied consisted of human fetuses at or near term, of new-born infants, and of isolated segments obtained from the adult at operation and at necropsy. A few studies were made on the dog and on the human *in vivo*.

The materials injected were the radio-opaque colloidal suspension of thorium dioxide, known as thorotrast; the vital dye brilliant benzo blue 6 B. A. extra conc., and the often used substances, air, mercury and Gerota's solution.

Studies in Vivo.—Based on the work of Hudack and McMaster[2] with the injection of vital dyes several studies were carried out. With the cooperation of Dr. I. S. Ravdin several injections, 0.1 cc. each, of expertly purified benzo blue 6 B. A. extra conc. were made intradermally into the closely shaved skin of the living dog; the areas used were the abdominal surface and the inner surface of the hind leg. The lymphatic collecting vessels filled rapidly and became clearly visible. Dye injected into the skin of the lower part of the abdomen and of the inner surfaces of the hind legs was carried quickly to the groin on the corresponding side. Lymphatic vessels arising from the skin of the upper part of the abdomen led to the axilla on the corresponding side. Three similar injections of benzo blue 6 B. A. extra conc. were made into the skin of normal humans, and several into the anal and peri-anal skin of patients. The results of these studies were valueless.

Because of these disappointing results the experiment was repeated on the skin of the dog. The lymphatics were again quickly and clearly demonstrated, and were readily followed for 10 cm. after nine to 15 seconds.

With a living female dog under amytal anesthesia, injections of benzo blue 6 B. A. extra conc. were made with the aid of a speculum, into the posterior vaginal wall at intervals from the introitus to, and including, the posterior lip of the cervix. The presacral nodes and the nodes at the bifurcation of the great vessels were clearly demonstrated within three minutes. Direct injections were made into the wall of the bladder, uterus, rectum, colon and small bowel; the lymphatics were visualized almost immediately.

Anatomic Study of the Lymphatics.—Air serves well for the demonstration of lymphatics, but the preparations are evanescent. The most satisfac-

tory results in this study were, however, obtained from the use of Gerota's solution and of mercury.

Thirty-five specimens (fetuses at, or near, term and new-born infants) were available for this research. Of this number, 14 were found satisfactory for study. The lymphatic drainage of the pelvic viscera, excluding the urinary tract, is summarized in Table I.

TABLE I

THE LYMPHATIC DRAINAGE OF THE PELVIC VISCERA, EXCLUSIVE OF THE URINARY TRACT

Viscera	Drainage Pedicles
Rectum	(1) Superior hemorrhoidal. (2) Middle hemorrhoidal (hypogastric).[5,7]
Anus	(1) Inferior group (inguinal).
Vagina	(1) Superior or external iliac. (2) Middle or hypogastric (3) Inferior { lateral sacral pedicle of sacral promontory.[5]
Cervix	(1) Anterior or external iliac. (2) Middle or hypogastric. (3) Posterior { lateral sacral. pedicle of sacral promontory.[5]
Uterus (and Adnexa)	(1) Lumbar. (2) External iliac. (3) Lateral sacral. (4) Inguinal.
Prostate	(1) Anterior or external iliac. (2) Middle or hypogastric. (3) Posterior { lateral sacral.[5] pedicle of sacral promontory.[5]

EXPERIMENTAL RESULTS

Anus.—Within the thickness of the anal and peri-anal skin is a lymphatic plexus of origin, the meshes of which are so finely patterned that a good mercurial injection resembles, in the cleared specimen, a solid layer of mercury (*b* in Fig. 1 *left*, and *d* in Fig. 1, *right*). More careful inspection of the plexus reveals the "nodular" appearance of the minute lymphatics resulting from the numerous valves within them and from overdistention by the injected mass.

From the lateral and anterior margins of the peri-anal plexus arise numerous larger collecting vessels which are among the afferent vessels of the inguinal nodes. These trunks, averaging eight to ten in number on each side, lying in the subcutaneous tissue, pursue a rather irregular course to the inguinal nodes (*c,c* in Fig. 1, *right*). Some of them lie in the genitocrural fold; others lie along the medial surface of the thigh. The collectors bifurcate and anastomose frequently along their course. The nodes (*d,d* in Fig. 2, *left*) appear as little tufts of mercury, giving rise in turn to efferent trunks which lead to the next nodal station. The efferents of the inguinal nodes lead to external iliac or to common iliac nodes (*e* in Fig. 2, *left*), the name depending on that portion of the

iliac vessels with which the nodal group is in relation. From the superior portion of the anal lymphatic plexus several small vessels can be seen to cross the anorectal line and to ascend along the rectal wall (*e* in Fig. 2, *right*). Hence there are definite anastomoses between the lymphatic plexuses of origin of the anal canal with those of the rectum.

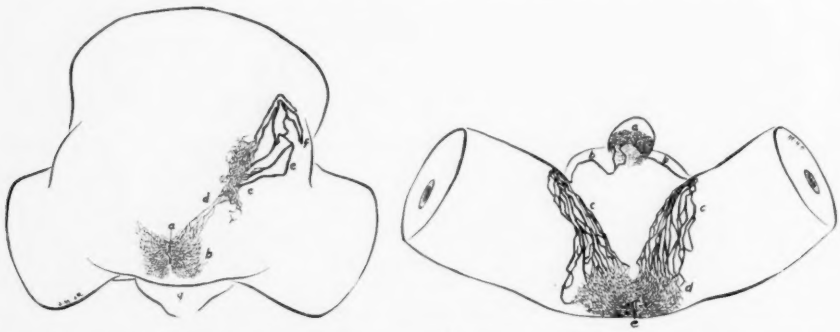

FIG. 1.—(*Left*) Peri-anal and gluteal lymphatics (posterior view) of a male, white fetus at term. Injections made along the anal margin and in the skin over the gluteal region, using mercury. (*a*) Anus. (*b*) Network of the peri-anal skin. (*c*) Plexus of the skin over the gluteal region. (*d*) Anastomotic vessels. (*e*) Collecting vessels. (*f*) Collecting vessels from the superior gluteal region passing to the groin. (*g*) Scrotum. (*Right*) Penile and peri-anal lymphatics, viewed from below, in the same specimen as that represented in Fig. 1 *left*. Injections made in the skin of the shaft, using mercury. (*a*) Network of the skin of the shaft of the penis. (*b,b*) Collecting vessels from the network of the shaft. (*c,c*) Collecting vessels from the peri-anal network. (*d*) Network of the peri-anal skin. (*e*) Anus.

Punctures made in the skin overlying the coccyx and lower part of the sacrum reveal a dense superficial network anastomosing freely with the peri-anal plexus, inferiorly, and with the superficial lumbar lymphatics or with the gluteal lymphatics, superiorly. Communications across the median line are plentiful (Fig. 3, *left*).

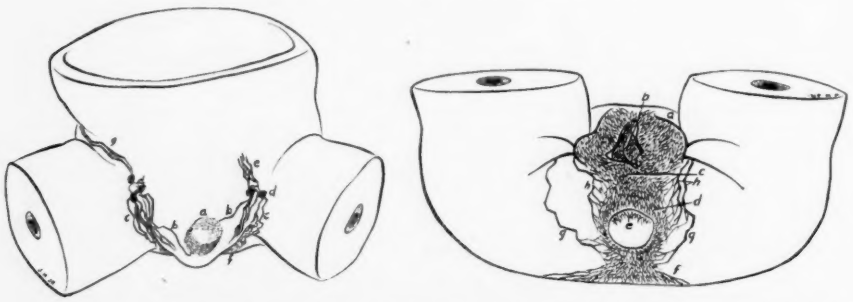

FIG. 2.—(*Left*) Lymphatics of the skin of the shaft of the penis (anterior view) in the same specimen as that represented in Fig. 1. Injection made in the skin of the shaft, using mercury. (*a*) Network of the skin of the shaft of the penis. (*b,b*) Collecting vessels from the network of the shaft. (*c,c*) Collecting vessels from the peri-anal network. (*d,d*) Inguinal nodes. (*e*) Beginning of the external iliac chain of the collecting vessels. (*f*) Network of the peri-anal skin. (*g*) Collecting vessels from the superior gluteal region. (*Right*) Peri-anal lymphatics and lymphatics of the external genitalia of a female, colored fetus at term viewed from below. The anus was dilated before fixation. Injections made in the skin overlying the lumbar, gluteal and sacrococcygeal regions, in the anal skin, in the skin of the fourchet and in the skin of the base of the right labium majus, using mercury. (*a*) Superficial lymphatic network of the labium majus. (*b*) Superficial network of the labium minus. (*c*) Superficial network of the fourchet. (*d*) Peri-anal network. (*e*) Anal network showing the origin of the vessels which ascend in the rectal columns of Morgagni. (*f*) Sacrococcygeal network. (*g,g*) Collecting vessels from the postanal plexus (sacrococcygeal). (*h,h*) Collecting trunks from the peri-anal and anal networks.

If the buttock be roughly divided into an upper and outer half and a lower and inner half, punctures along this line of division reveal two possible destinations for the injected material (Figs. 1, *left*, and 3, *left*). It may pass to the peri-anal plexus by way of the vessels of the lower and inner half of the buttock. Again, it may find its way upward

and outward, following the curve of the iliac crest, reaching the superficial external inguinal nodes (*c* in Fig. 3, *right*, and *g* in Fig. 2, *left*). Finally, as clearly shown in Figs. 1 *left* and 3 *left*, the material may fill both of these sets of vessels, thus completely encircling the limb.

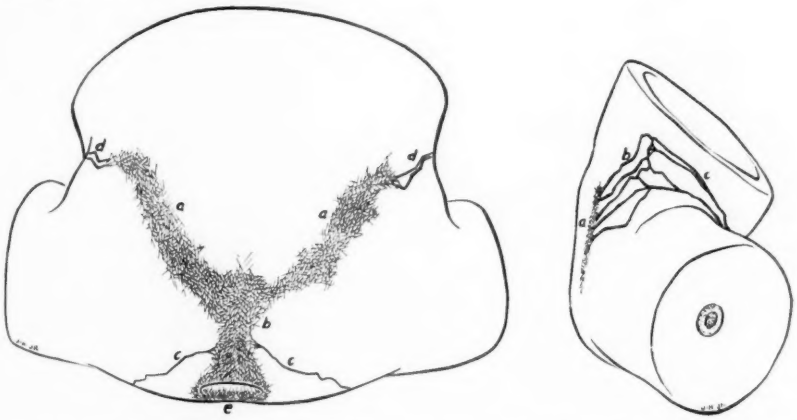

FIG. 3.—(*Left*) Anal and peri-anal lymphatics, and the lymphatics of the skin overlying the sacrococcygeal and the superior gluteal regions (posterior view) in the same specimen as that represented in Fig. 2 *right*. (*a,a*) Plexuses of the skin of the superior gluteal region. (*b*) Plexus of the skin overlying the sacrococcygeal region. (*c,c*) Collecting vessels from the sacrococcygeal plexus which pass to the groin. (*d,d*) Collecting vessels from the superior gluteal networks passing to the groin on either side by way of the superior route, following the curve of the iliac crest. (*e*) Anal network showing the origin of the vessels which ascend in the rectal columns of Morgagni. (*Right*) Lymphatics of the skin of the superior gluteal region (lateral view) in the same specimen as that shown in Fig. 1. Injections made in the skin over the gluteal region, using mercury. (*a*) Network of the skin over the superior gluteal region. (*b*) Collecting vessels. (*c*) Collecting trunks passing to the groin by way of the superior route, following the curve of the iliac crest.

Rectum.—In a coronal section of the rectum (Fig. 4, *left*) the anastomotic vessels which leave the anal plexus (*c*) to cross the anorectal line (*b*) and pass upward in the rectal wall, can be seen to lie within the columns of Morgagni (*a,a*). At the base of

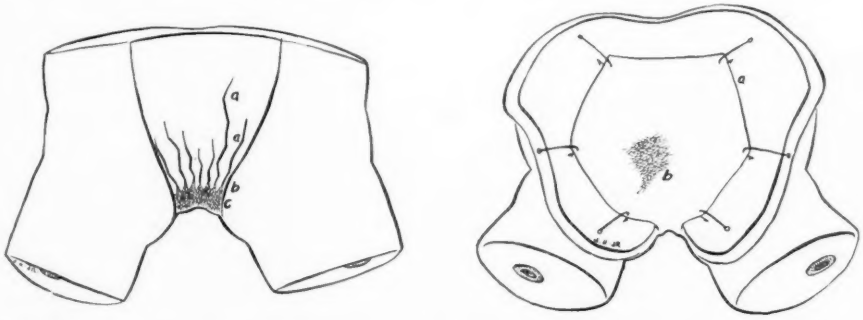

FIG. 4.—(*Left*) Coronal section of the rectum showing the anterior view of the posterior half of a male, colored fetus at term. Injection made in the skin of the anal canal, using mercury. (*a,a*) Lymphatic vessels lying in the columns of Morgagni. (*b*) Level of the anorectal line. (*c*) Network of the skin of the anal canal. (*Right*) Lymphatics of the rectal mucous membrane (anterior view) of a female, colored fetus at term. Injection made in the rectal mucous membrane, using Gerota's solution. (*a*) Rectum laid open. (*b*) Network of the rectal mucous membrane in the region of the upper portion of the ampulla.

each column (at the anorectal line) the anal lymphatics are gathered into a small tuft, from the apex of which one or two vessels ascend within the column for a distance of 1 to 3 cm.

The mucous membrane of the lower portion of the rectal ampulla does not possess

a well developed lymphatic plexus (Fig. 4, *left*, and 4, *right*). Above the columns, however, the rectal mucous membrane presents a rich network of lymphatics, although the demonstration of these vessels is difficult. Gerota's solution does not distend the lymphatics so well as does mercury. Hence a plexus can be more easily studied in a preparation in which Gerota's solution is employed (Fig. 5, *left*). The question arises as to

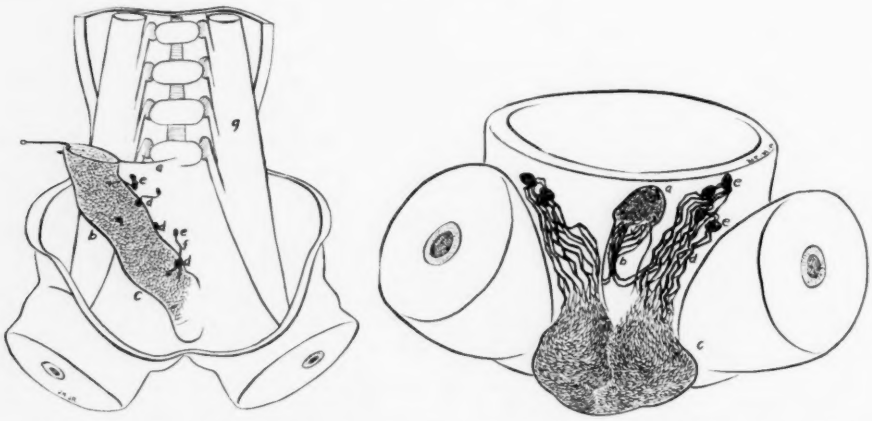

FIG. 5.—(*Left*) Lymphatics of the lower sigmoid and of the upper rectum (anterolateral view) of a male, white fetus at term. Injection made in the muscular layer of the gut, using Gerota's solution. (*a*) Cut edge of the mesosigmoid. (*b*) Network of the muscular layer of the sigmoid colon. (*c*) Network of the muscular layer of the rectum. (*d,d,d*) Pararectal nodes of Gerota. (*e,e*) Intermediate nodes lying in the mesentery. (*f*) Collecting vessel. (*g*) Psoas major muscle. (*Right*) Lymphatics of the glans penis and of the scrotum (anterior view) of a male, colored fetus at term. Injections made in the mucous membrane of the glans, and in the skin of the scrotum, using mercury. (*a*) Network of the mucous membrane of the glans. (*b*) Collecting vessels from the glans. (*c*) Network of the skin of the scrotum. (*d*) Collecting vessels from the glans and from the scrotum. (*e*) Superficial inguinal nodes.

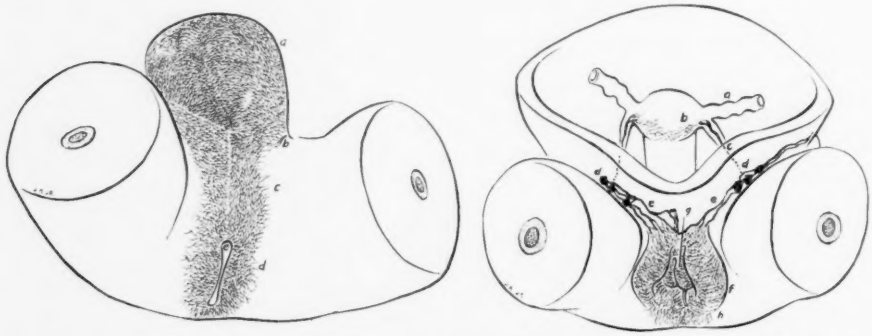

FIG. 6.—(*Left*) Scrotal, perineal and peri-anal lymphatics, viewed from below, of a male, colored fetus at term. Injections made in the peri-anal skin, in the skin of the perineum, and in the skin of the scrotum, using mercury. (*a*) Network of the skin of the scrotum. (*b*) Perineoscrotal collecting vessel. (*c*) Network of the skin of the perineum. (*d*) Network of the peri-anal skin. (*e*) Anus. (*Right*) Lymphatics of the external genitalia and of the round ligaments in the same specimen as that shown in Fig. 2 *right*. External genitalia injected as described for Fig. 2 *right*. Injections made also in the muscular layer of the uterine fundus, using mercury. (*a*) Fallopian tube. (*b*) Network of origin of the lymphatics of the round ligaments. (*c*) Collecting trunk of the round ligament passing to an inguinal node at *d*. (*d,d*) Inguinal nodes. (*e,e*) Collecting vessels from the superficial plexuses of the external genitalia. (*f*) Superficial network of the labium majus. (*g*) Superficial network of the labium minus. (*h*) Superficial plexus of the fourchet.

how this plexus can be distinguished from a vascular capillary network. In a gross study, such as this, the presence of the injected material in blood capillaries cannot be entirely eliminated. A deciding factor, however, is the successful injection of the collecting trunks, of the intermediate nodes, and of the regional nodes if possible. In Fig.

5, *left*, showing the lymphatic networks of the muscular layer of the rectum and of the lower part of the sigmoid, collecting vessels can be seen arising from the networks of origin at (*b*) and at (*c*) and passing to pararectal nodes (*d,d,d*) and to the nodes lying in the mesentery (*e,e*). These collecting trunks are the afferent vessels of nodes lying along the course of the inferior mesenteric artery. Some lie at the bifurcation of the superior hemorrhoidal artery, a few at the point of origin of the inferior sigmoid artery, a few at the origin of the left colic artery, and several at the root of the inferior mesenteric artery itself. The rectal lymphatic plexuses anastomose freely with those of the sigmoid at the rectosigmoid juncture.

Scrotum.—The rich lymphatic plexus of the scrotum is readily demonstrable by injections in, or near, the median raphé. One or two punctures usually suffice to fill the entire system. The collecting trunks arise near the raphé, swing laterally, receiving other collectors from the more lateral portions of the scrotal plexus, pass upward to the cruroscrotal fold, and finally end in the superficial inguinal nodes (*c*, *d*, and *e* in Fig. 5, *right*). The network of origin communicates posteriorly with the plexuses of the anus and perineum, laterally with those of the inner surface of the thighs, and anteriorly with that of the skin of the penis (Figs. 5, *right*, and 6, *left*).

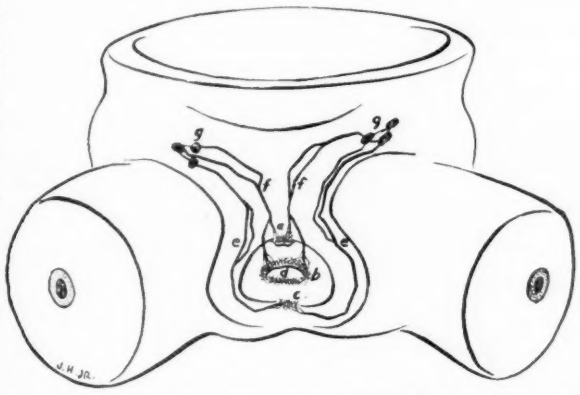

FIG. 7.—Deep lymphatics of the external genitalia of a female, colored fetus at term (anterior view). Injections made in the clitoris, in the skin of the vestibule, and in the skin of the fourchet, using mercury. (*a*) Network of the clitoris. (*b*) Superficial vestibular network surrounding (*d*) the vaginal introitus. (*c*) Superficial network of the fourchet. (*e,e*) Deep collecting vessels from the fourchet. (*f,f*) Deep collecting vessels from the networks of the vestibule and of the clitoris forming a coarse presymphyseal plexus. (*g,g*) Inguinal nodes.

Penis.—The lymphatics of the skin of the penis, like those of the scrotum, present a delicate plexus of origin along the raphé. Collecting vessels from this network pass around the penis to a pathway which lies superficially along its dorsum. Collecting trunks which drain the distal portion of the penis are obviously longer than those arising in the proximal portion. These vessels, on reaching the root of the organ, turn laterally to reach the superficial inguinal nodes of the corresponding side (*a,b,b*, Fig. 1, *right*, and *a,b,b*, Fig. 2, *left*). The proximal communications of the penile integumentary lymphatics are stated in the foregoing. Distally, these vessels anastomose with those of the prepuce, which in turn communicate with the lymphatics of the glans.

A single puncture in the glans penis is usually sufficient to demonstrate a very fine, dense plexus (*a* in Fig. 5, *right*). The collecting vessels gather on either side of the frenum and then swing dorsally, uniting as they do so, in three or four trunks which lie on either side of the deep dorsal vessels of the penis (*b* in Fig. 5, *right*). At the root of the organ they swing laterally, as do the other collectors, to the inguinal nodes on either side. The lymphatics of the glans anastomose with those of the distal portion of the urethral mucous membrane.

Vulva.—The lymphatic drainage of the external genitalia of the female is analogous to that of the external genitals of the male. Figs. 2 *right* and 6 *right* represent a specimen in which two punctures served to inject the entire system of the external female genital organs. One injection in the fourchet filled all the vessels of both labia minora, of the vestibule, of the clitoris, and of the left labium majus. One additional injection at the base of the right labium majus filled the remaining vessels. In this specimen the

plexuses of the labia majora (*a* in Fig. 2, *right*, and *f* in Fig. 6, *right*), are so dense that it is impossible to view the underlying collecting trunks which drain the fourchet, vestibule and clitoris. In Fig. 7 are shown the deeper trunks (*e,e*) which drain the area of the fourchet (*c*). These vessels, two on each side, lie in the subcutaneous tissue of the labium majus, the contour of which they follow in their course to the inguinal nodes. Medial to these collectors are the two deeper trunks (*f,f*) which arise, one on each side, from the upper portion of the vestibular plexus (*b*), and from the network of the clitoris (*a*). These collecting vessels enter into the formation of a coarse presymphyseal lymphatic plexus which, in some subjects, presents one or more small nodes. The free lymphatic anastomoses among the various parts of the external genitals is evident; one injection filled nearly the entire system on both sides, which is not an uncommon occurrence in the study of lymphatics.

Vagina.—Injection of the lymphatics of the vagina must be performed with as little dissection of the organ itself as possible. Removal of the anterior abdominal wall, of the anterior bony wall of the pelvis, and of the bladder and urethra provides good exposure of the vagina. The anterior vaginal wall is sectioned along the median line to the anterior lip of the uterine cervix. Punctures can then be made in the vaginal mucosa at any level. Again, intramuscular injections can be made along the posterior vaginal wall in the region of Douglas' cul-de-sac, with little or no dissection of the rectovaginal septum.

The vaginal lymphatic system presents two networks of origin, one mucosal and one muscular, which communicate with a perivaginal plexus giving rise, in turn, to the collecting vessels (Poirier and Cunéo[5]). The delicate mucosal network (*b* in Fig. 8) communicates inferiorly, at the introitus, with the vestibular vessels, and superiorly with the cervical vessels. In Fig. 8 are shown four collecting trunks arising from the left side of the perivaginal plexus. The two lower trunks (*h*) swing upward following a rather long but direct course to the lateral sacral nodes (*q*). The upper trunks (*i*) pass downward for a very short distance and then turn sharply upward. One of them ends in a lateral sacral node (*q*), the other turns laterally to reach a hypogastric node (*t*).

In Fig. 9 is shown the posterior perivaginal lymphatic plexus (*c*) which, together with the lymphatics of the anterior wall of the rectum (*d*), lies in the rectovaginal septum. The collecting vessels (*e*) of the vaginal network swing posteriorly, in the rectal stalks, to reach the lateral sacral node (*f*). One vessel (*i*) arises from the uppermost portion of the vaginal network, turns laterally, and ends in a node of the external iliac group (*j*).

Uterine Cervix.—The cervical lymphatic plexus of origin (*c* in Fig. 8) continuous below with that of the vagina (*b*), and above with that of the body of the uterus (*d*), is drained by collectors which ordinarily have two points of destination. The lowest cervical trunk (*k* in Fig. 8), dips downward for a short distance on the posterolateral surface of the vagina; it then turns upward to join the lateral sacral pedicle (*u*) of the vagina. The remaining cervical trunks in this preparation pass directly upward to reach the lateral sacral nodes, thus constituting the lateral sacral pedicle (*l*) of the cervix. In this same figure is shown an additional drainage pedicle. On the left side a cervical trunk of the upper group passes obliquely upward (*m*) in the broad ligament to join those vessels which constitute the lumbar pedicle (*r*) of the uterus. This is not a constant finding, having been noted but three times in 30 subjects by Cunéo[5] and Marcille. In Fig. 9 is shown a collecting trunk (*h*) of the external iliac pedicle, arising from the upper portion of the cervix and from the lower portion of the uterine body and passing laterally to end in nodes (*j*) which are in relation with the external iliac vessels.

Uterus.—The mucous, muscular and peritoneal networks of the uterus contribute to a subperitoneal plexus from which the collectors arise (Poirier and Cunéo[5]). This dense network, shown clearly in Fig. 8, *d* and *e*, is easily filled by random punctures in the muscular layer. The smaller collecting vessels arise near the median line on the anterior and posterior surfaces of the body of the uterus. They converge at the sides

of the uterus in such a manner as to form a "lymphatic ring." At *e* in Fig. 8, the fundus points directly toward the observer. The transparent preparation demonstrates this lymphatic ring clearly.

Collecting trunks from the lower portion of the body of the uterus may follow those of the cervix, which end in the nodes of the external iliac group (Fig. 9, *h*), or they may join the lateral sacral pedicle of the cervix as in Fig. 8, *l*. Thus the external iliac nodes may receive collecting vessels from the vagina, from the cervix and from the uterine body. The lateral sacral nodes may also receive afferents from the vagina, from the cervix, and from the body of the uterus.

One or two trunks arise from the anterior surface of the uterine fundus, just below the cornua, and accompany the round ligament on either side (*b* and *c* in Fig. 6,

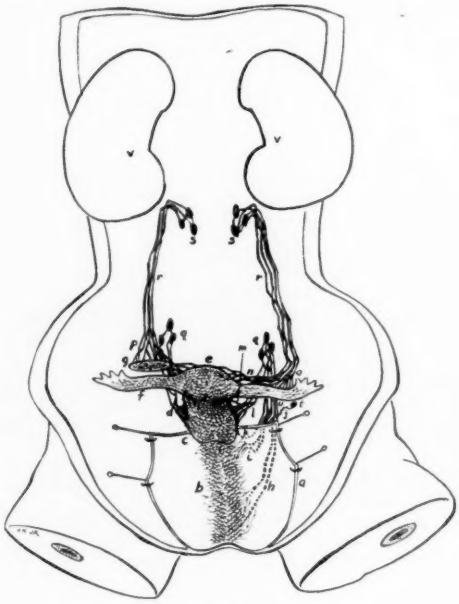

FIG. 8.

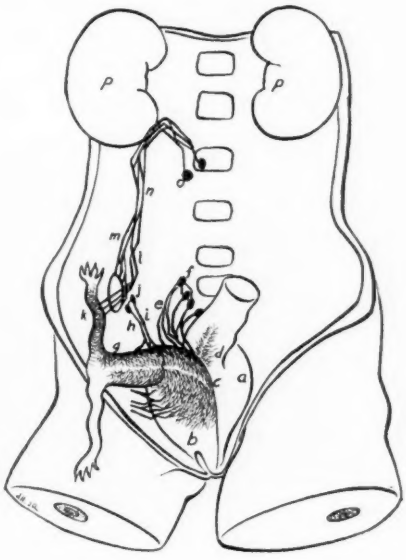

FIG. 9.

FIG. 8.—Lymphatics of the internal genitalia of a female, colored fetus at term (anterior view). Injections made in the mucous membrane of the vagina, posterior wall, in the muscular layer of the posterior lip of the cervix, in the muscular layer of the uterus and of the fallopian tubes, and in the ovaries, using Gerota's solution. (*a*) Vagina laid open to show the posterior wall. (*b*) Vaginal network. (*c*) Cervical network. (*d*) Network of the uterine body. (*e*) Network of the uterine fundus. (*f*) Network of the fallopian tube. (*g*) Network of the ovary. (*h*) Collecting vessels from the inferior portion of the vagina. (*i*) Collecting vessels from the superior portion of the vagina. (*j*) Collecting vessel from the vagina passing to a hypogastric node, *t*. (*k*) Cervical collecting vessel which joins vessels *h* and *i* to form the lateral sacral pedicle of the vagina passing to the lateral sacral nodes at *q*. (*l*) Cervical and uterine collecting vessels which make up the lateral sacral pedicle of the cervix passing to the lateral sacral nodes at *q*. (*m*) Cervical collecting vessel which passes upward to join the abdominal pedicle of the uterus. (*n*) Collecting vessels from the uterine fundus and from the upper portion of the uterine body entering into the abdominal or lumbar pedicle *r*. (*o*) Collecting vessels from the fallopian tube entering into the abdominal pedicle *r*. (*p*) Ovarian collecting vessels entering into the abdominal pedicle *r*. (*q,q*) Lateral sacral nodes. (*r,r*) Abdominal or lumbar pedicles. (*s,s*) Juxta-aortic nodes. (*t*) Hypogastric node. (*u*) Lateral sacral pedicle of the vagina. (*v,v*) Kidneys.

FIG. 9.—Lymphatics of the internal genitalia of a female, colored fetus at term (anterolateral view). The left broad ligament was sectioned near its lateral attachment, allowing the uterus to be partially rotated and displaced to the right. Thus the observer obtains a posterior view of the uterus and a posterolateral view of the vagina. The rectum remains in its normal position. Injections made in the muscular layer of the posterior vaginal wall, in the muscular layer of the cervix, of the uterus and of the fallopian tubes, and in the ovaries, using Gerota's solution. (*a*) Rectum. (*b*) Vagina. (*c*) Posterior perivaginal lymphatic network. (*d*) Muscular network of the anterior wall of the rectum. (*e*) Collecting vessels from the vaginal network. (*f*) Lateral sacral nodes. (*g*) Uterine network. (*h*) Collecting vessel of the external iliac pedicle, from the uterine body and cervix. (*i*) Collecting vessel of the external iliac pedicle, from the vagina. (*j*) External iliac nodes. (*k*) Network of the fallopian tube. (*l*) Collecting vessels from the fallopian tube entering into the abdominal or lumbar pedicle. (*m*) Ovarian collecting vessels entering into the abdominal pedicle. (*n*) Abdominal or lumbar pedicle. (*o*) Juxta-aortic nodes. (*p,p*) Kidneys.

right). The destination of these collectors is the inguinal group of nodes (*d* in Fig. 6, *right*).

The upper portion of the body of the uterus and the greater part of the fundus are drained by an abdominal or lumbar pedicle (*r* in Fig. 8). This group of collectors (*n* in Fig. 8) comprises from three to six vessels which swing laterally in the suspensory ligament of the ovary, and then upward, behind the ovary, accompanying the ovarian vessels. These lymphatic vessels lie on the anterior surface of the psoas muscle behind the peritoneum. In their upward course they cross the ureter, lying anterior to it. Opposite the lower pole of the kidney they bend medially at various angles to end in the juxta-aortic nodes (*s* in Fig. 8, and *o* in Fig. 9).

Ovary and Oviduct.—An injection made directly within the substance of the ovary (*g* in Fig. 8) usually serves to demonstrate the rich plexus of the hilum (*p* in Fig. 8).

The lymphatics of the fallopian tube (*f* in Fig. 8, and *k* in Fig. 9) are readily filled by means of a puncture near the horn of the uterus.

Collecting vessels from the ovary (*p* in Fig. 8, and *m* in Fig. 9) and from the tube (*o* in Fig. 8, and *l* in Fig. 9) join those of the lumbar pedicle (*r* in Fig. 8, and *n* in Fig. 9) of the uterus, the course and destination of which have been described in the foregoing.

Discussion.—This study indicates that the anorectal lymphatic collectors run in two pedicles, one inferior and one superior; the anorectal line is the boundary between the two networks of origin. The inferior pedicle carries vessels which end in the inguinal nodes. In the superior pedicle the lymphatics accompany the branches of, and the tributaries to, the superior hemorrhoidal vessels.

The present study does not demonstrate the additional components of the inferior pedicle of the anorectal lymphatics described by Rouvière[7] and by Villemin, Huard and Montagné.[9]

Quénu,[6] in 1893, described a middle hemorrhoidal ganglion (lymph node). This has been verified by Gerota, Cunéo, and Marcille and Boulay (Mondor[4]). However, Mascagni[3] indicated such a node in one of his illustrations.

The position of the middle hemorrhoidal node varies. It may lie along the course of the middle hemorrhoidal artery or it may be found at the point of origin of this vessel from the hypogastric artery. The afferent vessel or vessels of this node are pictured (Poirier and Cunéo[5]) as lying on the levator ani muscle and as arising in the region of the anal musculature. The present study yields no evidence, whatsoever, of the middle or hypogastric pedicle described by Poirier and Cunéo. Mondor[4] stated that the nodes of the middle pedicle are not often involved by metastases. Chlyvitch[1] expressed the belief that this pedicle is rare and that it is the least important of the three.

Mondor stressed the group of nodes situated at the bifurcation of the superior hemorrhoidal artery as being the "ganglion principal du cancer du rectum." He mentioned also the possibility of the occurrence of inguinal adenopathy in carcinoma high in the rectum, attributable to anastomoses between rectal and anal lymphatics. These connections were confirmed three times in the present study.

Sappey[8] has described the lymphatics of the anus and of the genitalia

very well, but made no mention either of rectal lymphatics or of the anastomoses between the lymphatics of the rectum and those of the anus.

The description offered here of the lymphatics of the external genitalia, in both sexes, does not differ in any essential points from those given in the literature on the subject. Likewise, the accepted descriptions of the lymphatics of the body and fundus of the uterus, of the fallopian tubes, and of the ovaries are confirmed by the results of the present research. However, with regard to the lymphatics of the vagina and of the uterine cervix, there are certain points which merit further discussion, as far as their relation to proctology is concerned.

Poirier and Cunéo described three groups of vaginal collecting vessels, a superior or external iliac pedicle, a middle or hypogastric pedicle and an inferior group which ends either in nodes of the sacral promontory or in a lateral sacral node. Rouvière[7] considered only two principal groups of vaginal collecting trunks, one accompanying the uterine artery and the other the vaginal artery. The former corresponds to the external iliac pedicle of Poirier and Cunéo, the latter to the hypogastric pedicle. Rouvière added that, rarely, vaginal collectors may reach the nodes of the promontory. The present study supports the existence of a lateral sacral pedicle and of a hypogastric pedicle. In none of the preparations was any evidence found of a vessel passing to the nodes of the promontory. However, it is shown that the lateral sacral group of collectors drains both the superior and the inferior portions of the vaginal tract. Furthermore, the hypogastric trunk is seen to arise from the upper third of the vagina rather than from the middle third. The external iliac pedicle is here confirmed as described in the accepted accounts. Rouvière believes that one cannot establish a definite distinction between the collectors arising from the different portions of the vagina.

The same three pedicles mentioned by Poirier and Cunéo in their description of the vaginal lymphatics were again mentioned by these authors in connection with the absorbents of the uterine cervix. Rouvière termed these groups anterior, hypogastric and posterior respectively, because of their relative positions within the pelvic cavity. The present study yields evidence of the anterior and of the posterior pedicles, but fails to demonstrate a hypogastric group. Rouvière stated that the disposition of the lymphatic collectors is subject to numerous and important variations.

As has been stated, the present study confirms the accepted descriptions of the lymphatics of the body and of the fundus of the uterus. This work also indicates the occasional existence of a lateral sacral uterine pedicle in addition to the lumbar, external iliac and inguinal pedicles.

The lack of evidence of the existence of certain groups of lymphatic vessels, however, does not necessarily mean their nonexistence. It does, however, indicate either the technical difficulty of injection or a difference in the anatomic structure of the material studied. Many of the specimens in this series were obtained from Afro-American subjects.

SUMMARY.—The lymphatics of the perineum and pelvis were studied *in vivo* and in anatomic specimens with particular reference to their relation to proctologic problems.

The study of the methods of demonstrating lymphatics *in vivo* is only partially complete. Of particular interest is the vital dye, brilliant benzo blue 6 B. A. extra conc., which was shown to be useful for the visualization and study of visceral lymphatics *in vivo*. This extends the work of Hudack and McMaster on cutaneous and subcutaneous lymphatics.

As to the studies on anatomic specimens, previous authors have covered the subject well, but none of them has had the more specific, clinical field of proctology in view. All of the previously described genito-anorectal lymphatic channels, with the exception of two, have been confirmed. As to those channels which could not be confirmed, namely the middle hemorrhoidal or hypogastric pedicle of the anorectal lymphatics and the pedicle of the sacral promontory receiving vessels from both the vagina and the cervix, it is not evident whether they were not found because of some fault in technic, or whether they were absent in the material examined. The fact that many specimens in this series were from subjects of Afro-American stock, which was not the case in earlier series, may be pertinent.

CONCLUSIONS

Of particular interest in this study in relation to present clinical problems, is the difference in the lymphatic drainage of the genital tract in the two sexes. In the male the lymphatic drainage from the genitalia is for the most part inguinal. In the female the lymphatic drainage from the external genitalia is inguinal, as in the male, but the drainage from the vagina and from the cervix is pelvic.

Of the pelvic lymphatics draining the female genital tract, the most important, from the standpoint of posterior spread of vaginal or of cervical infection, or of metastases from neoplasm, are those lying in the rectovaginal septum and those lying in the uterosacral ligaments or rectal stalks.

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TRANSURETHRAL RESECTION*

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THE idea of attempting to overcome obstructions at the bladder neck by means of transurethral manipulations is not new. The historical side of this subject has been presented by various authors during the past few years and those interested in this phase of the subject may refer to these various publications.[1, 2]

With the development of a new armamentarium for the non-surgical relief of bladder neck obstruction, widespread interest in electro-resection has been manifested both by the medical profession and the public. No surgical subject in urology, since the fulguration treatment of bladder tumors by Beer,[3] has received as much attention and discussion as has resection.

From a review of recent publications and discussions it is evident that definite ideas are beginning to crystallize regarding the selection of cases suitable for resection, its value and limitations, as well as the results obtained.

The statement seems justified that three different points of view exist today regarding the selection of cases suitable for resection.

(1) With the introduction of any new procedure in our armamentarium one finds a group of enthusiasts who take up the new procedure with great eagerness and in this particular instance believe that this method will replace, or even has already completely replaced, surgical prostatectomy.

(2) On the other hand, one finds another group who are just as decided in their views as the first group and who believe that surgical prostatectomy for various reasons is still the method of election.

(3) In the third group one finds urologists who are of the opinion that there is a definite place in our armamentarium for both transurethral resection and surgical prostatectomy. These men willingly admit without controversy that resection is the method of choice in the treatment of bars and contractures, small middle lobes, and, in some of the cases, of small lateral lobes, but that large hypertrophies should be treated by surgical prostatectomy. In other words, this group is of the opinion that one should make a rather careful selection of cases and not fit every patient to any one form of treatment.

It is at this point that there will naturally arise differences of opinion regarding the procedure to be employed in a given case. One surgeon may express his opinion that the prostate is too large to be resected and that a prostatectomy should be performed; whereas, another will state that resection is possible and will carry it out.

My experience in a relatively large number of patients has coincided with the latter opinion; and I think that the recommendation for resection or prostatectomy may be influenced by the personal preference of the surgeon.

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I have performed many resections, without difficulty of any kind, in patients who were told that this procedure was impossible. It is interesting to note the large number of doctors who come in for resection, despite the fact that they were told they needed open surgery.

On one point, however, there is very definite agreement, namely, in the treatment of carcinoma all agree that transurethral resection is the method of election combined with radium or deep roentgen therapy.

In trying to determine in my own mind the value and limitations of resection I have, as far as possible, approached this problem with an open mind and without prejudice.

My own position in this discussion can best be summarized by saying that during the past 51 months I have performed only one surgical prostatectomy and that during the same time I have refused resection to one patient only. This patient, a bad cardiac, was given suprapubic drainage until his cardiac function improved so that resection could be carried out. It is evident, therefore, that there has been no selection of cases, irrespective of the size of the prostate, the nature of the enlargement, or the physical condition of the patient.

From my experience in performing transurethral resections several very definite impressions have been gained; and while these views may not coincide with the views of others, I wish to present them at this time, realizing that perhaps some of you will not agree with me.

There is no doubt of the fact that the period of hospitalization is much shorter with resection than it is with surgical prostatectomy, and while this fact may not be of much importance to a small percentage of patients, I believe, all things being equal, it is an economic factor of enough importance to demand attention. Not only does this apply to the private patient, but also to the ward patient, as a result of which many more patients can be treated in a given period of time, hence making our ward services available to a larger number. The average stay in the hospital was seven days except when a preliminary suprapubic cystostomy was required, and in these cases the average stay was 20 days.

The number of patients in whom preliminary suprapubic cystostomy was employed became less and less as time went on. During the past three months only one preliminary cystostomy was done. On the other hand, I believe a cystostomy is indicated when catheter drainage fails because of chills, fever, pain, or bleeding, or when small stones with severe infection are present, or large stones.

Based on my experience with resection, I am convinced of the fact that resection has made it possible for a large number of patients to obtain relief from their prostatic condition who, because of serious coexisting disease in other important organs, were very poor surgical risks and, as a matter of fact, were refused prostatectomy elsewhere. It seems justifiable to state that if this procedure is of necessity the method of choice in the patient who is a very poor risk and hence cannot undergo a surgical prostatectomy with safety, the assumption that it should be made available to every patient who is a good risk is unquestionable.

It is not an uncommon experience to have a patient sent in by his physician with the request that a resection be performed because the patient is not a good risk for prostatectomy. The following instance is a good example of this type of case.

Case 1.—A. G., aged 76, admitted to the Presbyterian Hospital, March 1, 1936.

Present Complaint.—Frequency of urination for five years with gradually increasing severity; nocturia that has gradually increased, for three years. The day and night frequency accompanied with a great deal of pain on urination. During the past year there has been a good deal of difficulty in starting the stream; it was necessary to strain a long time before the urine would flow. Urgency and imperative urination during the past three weeks.

Physical Examination.—Pupils equal and reacted somewhat slowly to light. Numerous coarse and squeaking râles in the lungs; breath sounds at both bases tubular in character. The apex heart beat was not located; the sounds, of very poor quality. Abdomen: marked diastasis of the recti and a double inguinal hernia. Bladder dulness reached to the umbilicus. Liver palpable one inch below the costal arch. Rectal examination: a three plus enlargement of the prostate that was smooth and firm. Seminal vesicles could not be felt. The edema of the feet and ankles and legs extended almost to the head of the fibula. Lips and finger tips, cyanotic.

Laboratory Data.—Blood: red cells, 4,250,000; white cells, 10,800; hemoglobin, 74 per cent. Blood pressure: systolic, 170; diastolic, 70. Urine: albumin, 3 Mm.; sugar, 0; blood, +; leukocytes, 5,000. The colon bacillus was cultured from the urine. Blood chemistry: urea nitrogen, 14.3; creatinine, 1.2; nonprotein nitrogen, 33.2.

Roentgenologic Examination.—A marked diverticulosis of the colon and a bilateral hernia of the diaphragm. Multiple stones in the bladder. A small, contracted bladder with multiple cellules and small diverticula in the cystogram.

Indwelling catheter drainage was carried out for eight days.

Diagnosis.—Bilateral diaphragmatic hernia; diverticulosis of colon; bronchial asthma and emphysema; bundle branch block; myocardial damage; hypertension; arteriosclerosis; benign hypertrophy of the prostate; vesical calculi; chronic cystitis; bilateral inguinal hernia; diverticula of bladder, and hydrocele.

Operations.—On March 9, 1936, under sacral anesthesia, a transurethral resection was performed and a very large middle lobe and two rather large lateral lobes were removed. On March 16, 1936, under sacral anesthesia, a litholapaxy was performed. The patient had an uneventful convalescence. Discharged from the hospital, March 22, 1936.

Another interesting observation noted was the fact that the number of cardiac cases seeking relief showed a very definite increase. Whereas, in the days of surgical prostatectomy about 35.8 per cent of the patients suffered from cardiovascular lesions, a recent study of my resection cases showed 64.3 per cent, an increase of 28.5 per cent. This may be due to the fact that a more careful study of each case was made and hence more heart lesions were found than formerly. An electrocardiogram was made in each case prior to resection, and furthermore there is no doubt in my mind of the fact that more cardiacs were sent in for resection than were sent in for open prostatectomy because of the lower mortality rate.

Many of the patients with hypertension have been sent in with the request that a resection and not a prostatectomy be performed. The highest systolic pressure found was 260, and a resection was performed without the slightest difficulty.

The following case is presented as an example of a patient with a severe heart lesion.

Case 2.—M. C., aged 75, admitted to the Presbyterian Hospital, November 26, 1935.

Present Complaint.—Complete retention for ten days during which time he has been catheterized three times a day. There is some mental confusion.

Physical Examination.—A fairly well preserved elderly man. Head and neck, negative. Lungs, considerable wheezing at the left base posteriorly and moist râles. Heart, enlarged; irregular in rhythm; the sounds of poor quality; pulse, that of an auricular fibrillation. Abdomen, negative. Rectal examination: a two plus enlargement of the prostate that was smooth and regular.

Laboratory Data.—Blood: red cells, 4,350,000; white cells, 7,810; hemoglobin, 88 per cent. Blood pressure: systolic, 142; diastolic, 60. Urine: sugar, 0; albumin, 0; blood, 0; red blood cells, 0; white blood cells, 140; casts, 0. Blood chemistry: urea nitrogen, 14.8; creatinine, 1.4; nonprotein nitrogen, 32. Phthalein test: a total of 62 per cent of the dye eliminated in one and one-half hours.

Electrocardiogram.—Auricular flutter; ventricular extrasystoles; myocardial damage.

Roentgenologic Examination.—Negative for stone. A small bladder with trabeculated margin and some out-pouching from the left toward the base in the cystogram.

After three weeks of indwelling catheter drainage, bed rest and heart medication, the patient showed great improvement.

Diagnosis.—Coronary disease; auricular fibrillation and flutter; cardiac decompensation; cardiac hypertrophy and dilatation; myocardial damage; chronic bronchitis, and benign prostatic hypertrophy.

Operation.—On December 19, 1935, under sacral anesthesia, a transurethral resection was performed. The patient developed postoperative bleeding on January 6, 1936, three weeks after the resection. Two bleeding vessels were found; they were fulgurated and the bleeding promptly stopped. Patient discharged from the hospital January 17, 1936.

The importance of a careful study of the renal function requires no emphasis at this time, nor is it necessary to call attention to the fact that resection should never be performed until the renal function, if impaired, has been restored to normal or at least until it has become stabilized.

In the largest number of cases the improvement in renal function with treatment is rapid, as judged by the results of the functional tests as well as by the patient's general condition. In this group of patients the problem of determining the most opportune time for resection offers no serious problem.

I wish, however, to discuss the group of cases with badly impaired renal function in which the response to treatment was very slow and the improvement hardly perceptible, and the functional tests remained fixed at a high, or relatively high, level. At times these levels were very high so that the danger of renal insufficiency was an opposing factor in case the patient was to be operated upon. It is in this group that the decision to operate or not to operate is very difficult and at times greatly taxes one's clinical judgment. There is no doubt in my mind that in just this type of case transurethral resection should be the operation of choice and certainly carries with it less risk than does surgical prostatectomy. As an illustration of this type the following case is presented.

Case 3.—C. E., aged 57, referred by Dr. J. B. Herrick, was admitted to the Presbyterian Hospital, January 10, 1936.

Present Complaint.—Frequency of urination, urgency, burning, dribbling, and noc-

turia. These symptoms had been present for ten years. Patient has had pain in the epigastrium for ten years, nausea and vomiting for several months, and very tarry stools for several days.

Physical Examination.—Moderately well nourished; exceedingly pale and sallow. Head, neck and lungs, negative. Heart enlarged to the left; a systolic murmur at the apex. Examination of the abdomen showed a large tumor mass extending four finger-breadth above the umbilicus. Liver, palpable. Rectal examination: a one plus enlargement of the prostate, smooth, with no signs of carcinoma.

Laboratory Data.—Blood: red cells, 2,520,000; leukocytes, 9,850; hemoglobin, 44 per cent. Blood pressure: systolic, 150; diastolic, 96. Urine: albumin, 0; sugar, 0; blood, 0; leukocytes, 0. Blood in the stool. Blood chemistry: urea nitrogen, 105.6; creatinine, 6.3. Phthalein test: only a trace of dye excreted in one and one-half hours (Table I).

Roentgenologic Examination.—Negative for stone. A set of intravenous pyelograms showed no visualization. Further studies by Doctor Herrick established the fact that the patient had a duodenal ulcer and a polyp in the pyloric antrum.

A catheter was passed into the bladder and gradual decompression instituted. This required 21 days. The patient was given three blood transfusions, each 500 cc. of whole blood.

TABLE I

SUMMARY OF THE FUNCTIONAL TESTS IN CASE 3

				Phthalein Test	
Date	Urea N.	Creatinine	N.P.N.	Appearance Time	% in 1½ hrs.
1-11-36	105.6	6.3	.....		
1-13				45 min.	Trace
1-16	69.8	3.7	74.4		
1-23	83.3	2.9	102.6	42 min.	Trace
1-29	102.5	2.6	123.0		
2- 6	129.0	2.9	142.9		
2-12	91.8	2.7	125.8		
2-17	90.1	2.9	105.3	55 min.	Trace
2-24	58.8	2.7	74.1		
3- 2	55.1	2.5	86.7		
3- 4	55.6	2.5	72.7		
3- 8				10 min.	11%
3- 9	56.2	2.3	92.2		
3-18	59.2	2.4	76.2		
3-21				19 min.	30%
3-23	54.0	2.9	70.2		
3-30	54.4	2.6	61.5		
3-31				12 min.	26%

Diagnosis.—Polyp of stomach; ulcer of duodenum; marked secondary anemia; hypertension; severe hemorrhage from intestinal tract; benign prostatic hypertrophy; chronic urinary retention; marked renal insufficiency; bilateral hydronephrotic atrophy of kidneys.

Operation.—On April 1, 1936, a transurethral resection, under sacral anesthesia, was performed. The patient had an uneventful convalescence. Discharged from the hospital on the fifteenth postoperative day.

I cannot agree with those who believe that because resection carries with it a much lower mortality rate, it is a relatively simple procedure, and that careful, detailed preoperative study and preparation can be dispensed with

and resection instituted at once. It seems to me that this is both poor teaching and poor practice. Because of the large number of cases that I have seen with serious organic disease,[4] not only in the upper urinary tract but in other organs, I believe it is absolutely necessary to obtain a careful history, as well as make a complete physical examination in each case and carry out the necessary preoperative treatment.

As my experience with resection has widened I have operated upon a certain group of cases without preliminary catheter drainage. Heretofore all patients who had four ounces or more of residual urine were given preliminary catheter drainage. At the present time no preliminary drainage is em-

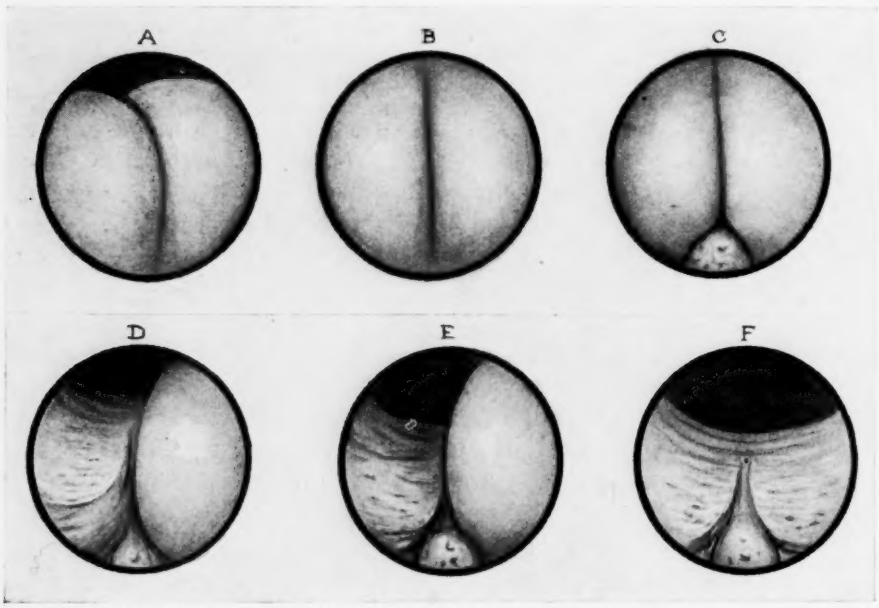

FIG. 1.—(A) Showing a small view of the bladder cavity and the presence of two large lateral lobes. (B) Showing the presence of the lateral lobes and the very markedly narrowed urethra. (C) Showing the lateral lobes and the verumontanum. (D) Partial removal of the right lobe. (E) Complete removal of the right lobe. (F) Complete removal of both lobes. The verumontanum is intact.

ployed in a patient in whom the residual urine does not exceed six ounces, provided the urine is clear, the renal function normal, and the heart and lungs are in good condition.

Preliminary cystoscopy is no longer carried out as a routine; once the diagnosis of prostatic obstruction has been made the type of enlargement is determined at the time the resection is performed. The exception to this rule is in case the history is not typical for prostatic obstruction, there is a history of one or more attacks of hematuria, and the cystogram shows a filling defect.

After the sheath has been passed into the bladder the examining telescope is introduced and the bladder cavity examined for tumors and stones. A search for diverticula is also made and finally the configuration of the bladder orifice is determined (Fig. 1 A, B and C).

The bladder orifice is next examined with the retrograde lens, and a good deal of information is obtained regarding the size of middle lobe and bars (Figs. 2 A and B and 3 A). Hypertrophy of the interureteric ligament and the resulting pouch behind it can best be demonstrated with the retrograde lens. When these conditions are marked I always resect the hypertrophied interureteric ligament.

The third and last step is the examination of the prostatic urethra to determine the size and extent of the intra-urethral enlargement and its relationship to the verumontanum (Fig. 1 C).

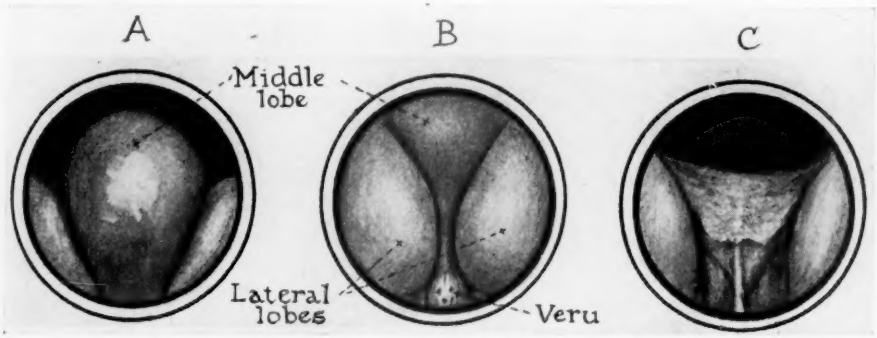

FIG. 2.—(A) Showing the presence of a large middle lobe. (B) View showing the lateral lobes, the middle lobe, and the verumontanum. (C) Beginning resection of the middle lobe.

POSTOPERATIVE REACTIONS.—The general opinion seems to prevail that following resection there are fewer postoperative complications and that when they do occur they are not so severe and are relatively much shorter in duration. I can subscribe to these views.

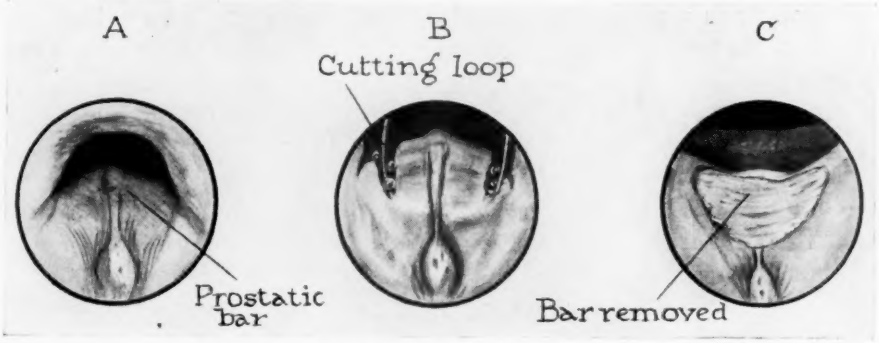

FIG. 3.—(A) Showing the presence of a bar. (B) Removal of bar. (C) The hypertrophied interureteric ligament comes into view.

MULTIPLE RESECTIONS.—Objections to this method have been made because of the fact that a second or even a third resection is necessary. This, I do not believe, is sound reasoning and requires some qualification. In this series of resections a second one was performed in 10.6 per cent of the cases, and a third one in 3.7 per cent. A study of this group shows that the cases can be divided into, and discussed under, three headings.

(1) In the cases of carcinoma a second resection is necessary when the carcinoma, because of its recurrence or continued growth after the resection, again produces obstruction. I believe that two resections for carcinoma are preferable to one suprapubic cystostomy and a permanent suprapubic drain or catheter.

(2) In this group are the patients with large benign hypertrophies in whom, it can readily be understood, the obstruction cannot be removed at one operation, because of the size of the growth. I always tell these patients that a second resection may be necessary.

(3) In the remaining cases in this group are the patients in whom not enough tissue was removed at the first resection. This occurred with some frequency in the early cases, but with experience a second resection became less and less frequently necessary.

HEMORRHAGE.—Before the technic of resection was completely mastered, hemorrhage at the time of operation was at times a serious problem, but with experience this untoward occurrence becomes less and less of a problem, although occasionally a serious hemorrhage occurs no matter how careful one may be. Careful search for the bleeding point should be made and its immediate control effected. I endeavor to have the patient return to his room with the wash water free of blood, that is, clear or faintly tinged. Each bleeding point should be isolated and fulgurated before proceeding with the resection, and, above all, blind indiscriminate coagulation is to be avoided. I believe that late secondary bleeding and the persistence of infection are due in some cases to indiscriminate widespread and deep coagulation.

Secondary bleeding occurring on the tenth to the fourteenth day was present in 19 cases but was never severe. Clots, when present, were evacuated with the Bigelow pump and the bladder washed out with a warm solution of silver nitrate. In most of the cases this controlled the bleeding, but when this procedure failed the resectoscope was introduced, and the bleeding point fulgurated or excised.

Late hemorrhage occurred in 14 cases, at periods varying from three months to two years after the resection. In the cases that I have had an occasion to reëxamine, enlarged vessels around the bladder orifice and in the prostatic urethra were found. These were destroyed by fulguration. An interesting fact is that these late hemorrhages occurred in patients with very large prostates.

In one instance in a patient aged 80, hemorrhage occurred 26 months after resection. A suprapubic cystostomy was performed (elsewhere) in order to control the bleeding. The patient was unable to withstand even this simple procedure and died.

EPIDIDYMITIS.—The incidence of epididymitis has diminished as experience with resection has grown. Of the first 117 cases 15 per cent had this complication, and a routine vasectomy was done, an operation no longer employed, as this complication is very rare.

RESULTS.—In order to determine the results obtained with resection, two methods of investigation were used: (1) In so far as it was possible, every

attempt was made to have the patients return to the office or hospital for a personal examination. (2) Many of the patients who lived out of town could not, or would not, travel to the city for personal interviews and examinations, hence it was necessary to resort to a questionnaire.

When the obstruction had been completely relieved and there were no complicating conditions present, the symptoms rapidly subsided, the pyuria disappeared, and the urine became clear. It might be well, at this time, to mention the fact that in some of the patients in whom the pyuria persisted, without symptoms and without residual urine, and in whom no local treatment was instituted, examination at the end of from three to six months

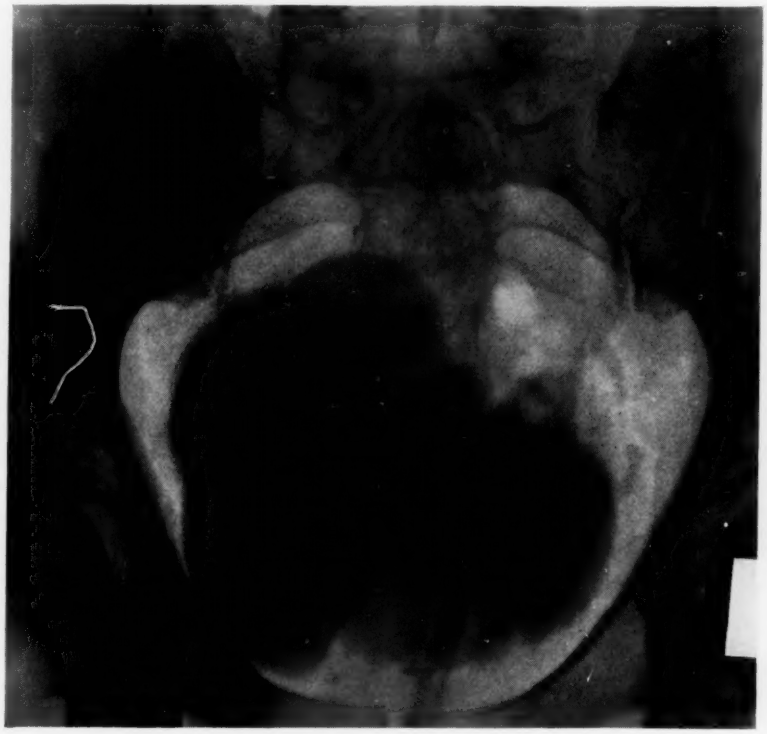

FIG. 4.—Showing a diverticulum much larger than the bladder.

showed a complete disappearance of the pyuria. In other words, in some of the cases a certain elapse of time was necessary before the urine became clear. This can readily be understood because cystoscopic examination in some of these cases still showed the presence of white sloughs at the end of 11 weeks. Aside from the slow healing in some of the cases there were other causes for the persistence of the pyuria, among which may be mentioned:

(1) *The Presence of One or More Diverticula.*—In some cases one or more diverticula were responsible for the turbid urine, although the patient was symptom free; under these circumstances it was difficult to convince the patient that a diverticulectomy should be performed in order to clear up the turbid urine (Fig. 4).

(2) *The Presence of Large Hydronephrosis.*—In some of the cases of persistent pyuria, hydronephrosis was the contributing factor; even after the prostatic obstruction had been completely relieved, the hydronephrosis and hydro-ureter persisted, with stasis and infection, and hence pyuria. This cannot be reasonably charged as a failure of resection.

(3) *Infection in the Seminal Vesicles.*—Infection in the seminal vesicles as a cause of the persistence of symptoms and turbid urine following surgical prostatectomy is recognized by all urologists, and was stressed by Boyd[5] some years ago. If symptoms and turbid urine persist after a reasonable time and if infection is found in the seminal vesicles upon examination, then the use of heat per rectum, systematic massage, and instillations will result in completely alleviating the symptoms and clearing up the urine. It is well to bear in mind that even when the urine is clear infection in the vesicles may be the cause of symptoms.

(4) *The Formation of Stone.*—Calculus formation following resection has been rare in my experience, having occurred only four times; three in my own cases and once in a case resected elsewhere. After litholapaxy there was a complete disappearance of the pyuria and the symptoms.

(5) *Failure to Relieve the Obstruction.*—Naturally, failure to relieve the obstruction results in a continuation of the symptoms, as well as a persistence of the pyuria.

(6) *Indiscriminate, Widespread Coagulation in Controlling the Bleeding at the Time of Resection.*—This has already been considered under hemorrhage.

Residual Urine.—The objective in the treatment of the patient suffering from prostatic obstruction has, as its ultimate goal, the relief of the obstruction; whether or not this result has been attained can best be determined by the estimation of the residual urine. It is necessary to bear in mind that the amount of residual in a certain number of cases will gradually diminish. I have seen patients who, when discharged, still had an ounce or two of residual urine, and when examined six months later had only a few cubic centimeters. It is fair to assume, therefore, that in some of the patients mentioned in the table, smaller amounts of residual urine could be demonstrated if the opportunity of an examination were presented today.

I have had the opportunity of examining 273 patients for the purpose of determining the question of residual urine (Table II).

Table II

AMOUNTS OF RESIDUAL URINE IN 273 CASES FOLLOWING TRANSURETHRAL RESECTION

No residual urine	181 cases	(66.30%)
10–20 cc.	44 cases	(16.11%)
Total		(82.41%)
20–30 cc.	17 cases	(6.22%)
30–40 cc.	5 cases	(1.83%)
40–50 cc.	6 cases	(2.19%)
50 or more cc.	20 cases	(7.32%)

A detailed analysis of the 20 patients who had 50 or more cubic centimeters of residual urine is presented in Table III.

TABLE III

ANALYSIS OF CASES WITH HIGH RESIDUAL URINE

Diverticula of bladder	5 cases
Carcinoma of prostate	5 cases
Large hydronephrosis	2 cases
Bladder and prostatic calculi	2 cases
Periprostatitis	1 case
Second resection advised	2 cases
No return for check-up	3 cases

In some of these cases the persistence of turbid urine was due to changes which might be directly ascribed to the bladder neck obstruction and hence one cannot rightfully classify such cases as failures; for instance, in five cases the diverticulum was responsible for the persistence of turbid urine, and I am of the opinion that part of the residual urine can also be explained on this basis. In the cases of carcinoma of the prostate the carcinoma continued to grow and hence produced obstruction. In two cases the large hydronephrosis was responsible for turbid urine and a certain amount of residual. The two patients with bladder calculi were relieved of the turbidity of their urine and obstruction as previously mentioned under the subject of formation of stone following resection. In two cases sufficient tissue had obviously not been removed and the patient refused to have a further resection. And, finally, in three instances it was impossible to have the patients return for a check-up.

INCONTINENCE.—There has been no instance of complete incontinence in this series of 804 resections. Shortly after leaving the hospital a small number have had difficulty in holding urine. This was not due to incontinence, but to urgency and imperative urination. Continence was eventually recovered. In a few instances the patients would lose a few drops, but this was temporary and, as the infection cleared up, the condition rapidly disappeared. In two instances the patients' loss of a few drops was due, not to incontinence, but to stricture of the urethra.

In one instance, a recent case, lack of control was rather marked and annoying during the daytime, but at night the control was perfect. The patient was given ephedrine, and at examination, three days ago, showed great improvement.

In a second case, when last seen three months ago, the patient had perfect control at night and during the day except when he assumed certain positions, and then a few drops would escape.

URETHRAL STRICTURE.—Whether or not stricture formation will occur in a sufficiently large number of cases, to justify the statement that resection produces stricture, must be determined with the passage of time, and the same should be said regarding the question of whether or not it will occur more frequently with resection than it did with surgical prostatectomy. Doubt has recently been expressed as to whether or not resection produces a stricture,

it being the opinion of some that when stricture is found following resection the stricture was present before operation.

Sexual Function.—So far as the sexual function was concerned, there has been no decided change. In the majority of the cases there has been no change, whereas, in some, with an improvement in the general condition, there has been an improvement in the sexual function. In only a few cases has there been a slight impairment of function.

Recurrence.—A good deal of discussion has centered around the question of whether or not recurrence will take place following transurethral resection. This question cannot be definitely answered at this time. It is a well known fact that recurrences have taken place following both suprapubic and perineal prostatectomy. One of the characteristics of recurrence following surgical prostatectomy is the fact that the interval between the removal of the prostate and the development of symptoms which demand further prostatectomy averages about eleven and one-half years.[6] It stands to reason that if recurrences are going to occur, the time since the introduction of resection has not been long enough for them to develop. On the other hand, should recurrences occur after a period of five or ten years, a second transurethral resection should be a perfectly simple procedure.

Mortality.—The mortality rate has fluctuated from time to time, depending in part upon the type of cases and whether or not one wishes to refuse resection to many of the so called "bad risk" cases, which I have not done. My records show that I have performed 184 resections with but one death—a mortality of .54 per cent. Recently a large number of "bad risk" cases have presented themselves and the mortality in 804 resections was 3.9 per cent.

If resection can relieve both obstruction and symptoms, and also has to its credit a shorter period of hospitalization, a lower mortality rate, and fewer postoperative complications than in the cases in which prostatectomy is performed, it obviously has merit and should find its proper place as a surgical procedure.

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Discussion.—Dr. Edwin Beer (New York, N. Y.).—A certain amount of exaggeration and overenthusiasm may be necessary to call attention to, and to put over, a new surgical procedure. Whether this is due to the natural lethargy of a large part of the medical profession, I do not know. As a result of this overswing of the pendulum, at times harm is done, but this is usually only a temporary state. Eventually the pendulum swings back, a normal equilibrium is established, and the exact position of such a new procedure as Doctor Kretschmer has so clearly presented to us, is recognized. This cor-

rection of the overenthusiasm is already manifest in the literature, and before long we shall recognize clearly the possibilities of this new method of attack, and at the same time its limitations and dangers.

About 1909, Hugh Young started the transurethral removal of fibrous obstructions at the bladder neck, after the various attempts of European surgeons along these lines, during the latter half of the last century, had given way to the operation of prostatectomy. One year later the profession became acquainted with the surgical use of the high frequency current in the bladder, and its possible therapeutic value in instances of bladder papillomata, tuberculous ulcers of the bladder and prostatic adenomata. Since those days, the instrumental advances and the modalities of the currents have been much improved and modified, so that at present we have a variety of instruments for cutting out pieces of the bladder neck and of the prostate, either by punch procedures followed by cauterization, or by resectoscopes with loops of one sort or another, activated by the high frequency cutting current of such oscillations and intensity as to cut living tissue under water. These changes are in the nature of a great and fundamental advance.

The three types of cases in which these new methods are of use are as follows:

(1) Contractures of the neck, which in years past were excised by wedge excision from above through the bladder. These can be cut with a point or loop electrode with complete relief of symptoms, using an ordinary operating cystoscope or a resectoscope. This is a great improvement over the cold punch operation or the suprapubic excision. The latter procedure or approach, however, may be useful when the exact physical status of the neck of the bladder is not accurately visualized.

(2) Obstructions caused by carcinoma of the prostate also lend themselves very satisfactorily to these transurethral resections, which may be repeated as new obstructions develop. The pathologic tissue removed during these operations is often helpful in establishing the correct diagnosis.

(3) When it comes to the ordinary and usual type of prostatic hypertrophy or adenoma, the field of transurethral resections has no limitations for some enthusiasts and very definite limitations for others. In this group of patients, there is a subgroup with mild symptoms of frequency and questionable obstruction, associated with a very small amount of residual urine and a very moderately enlarged prostate, upon whom no experienced surgeon would have felt justified in performing a prostatectomy. These patients present a definite potential field for transurethral resections, and may represent a large proportion of the total in most published series of resections. The frequency, and perhaps infection, do not regularly disappear, but may be made worse following these excisions of some prostatic tissue, despite reduction of residual urine. These cases are definite beginning prostatic adenomata and have nothing to do with those cases which have been resected prophylactically before any definite complaint has developed. It is doubtful whether resection affords these patients the expected relief with any regularity. If the subjective complaints are well developed, one is justified in attempting to give relief by resection, even though it may be only temporary and incomplete. Correct diagnosis here, as always, is essential.

In addition to these mild prostatics who frequently are worse off after a resection, there are two other groups of definite prostatic adenomata that must be discussed: (a) the moderately large, bilobe, trilobe or single lobe prostate, and (b) the very large, more or less trilobe or bilobe hypertrophy. In those localities where no pressure is brought to bear upon the surgeon by competitors or by the patient or his friends, the opinion is gradually devel-

oping that the last mentioned group of very large, succulent adenomata should be treated radically and definitively by prostatectomy or enucleation of the adenomata, either by the suprapublic or the perineal approach. This attitude seems to be based on a great many factors, without denying that if the "resectionist" is patient he can whittle away a fair amount of the largest prostates in one or more sessions.

The operation of suprapubic prostatectomy in two stages has gradually assumed the status of an almost minor procedure, with a low mortality, 6 plus per cent, usually incidental to cardiovascular disturbances, and with complete cure and no chance of recurrence, if properly performed. On the other hand, the transurethral approach in these cases frequently is followed by a whole group of new complications, such as primary or secondary hemorrhage into the bladder, requiring repeated evacuation of clots, invariably causing great discomfort, infection of the bladder and the more or less completely resected prostate base, which frequently, much more frequently than is reported, may turn the scales against the patient, not to mention the various instrumental damages and perforations that are reported by competent instrumentalists. In this group of cases, the prolongation of the necessary instrumental work and the various complications are liable to have a definitely higher mortality than follows a two stage suprapubic enucleation. This is our experience, and I believe if this type of case were alone considered, the data of other clinics would probably bear me out. The trouble with most statistical studies along these lines is that the mortality statistics are grouped covering the simple cases of contracture, the small, almost symptomless hypertrophies, the prophylactic excisions, perhaps incorrectly interpreted cystoscopically, and the moderate sized but definite prostatic adenomata, as well as the large hypertrophies.

In the moderately enlarged prostates, as opposed to the very large adenomata which weigh one or more ounces and are as large as one to three or more lemons, there is a definite field of usefulness for this method, even though repeated sessions may be required before the patient can void and be comfortable, and even though one may, as time goes on, see more and more of these partially removed adenomata redevelop and produce recurring symptoms, which is a rare sequela to a properly executed, complete enucleation. There is no doubt that in this group of moderately enlarged prostates one is justified, if the patient requests it, and especially if he is a poor surgical risk owing to poor renal function, coronary disease, diabetes, and so forth, in using transurethral resection. From my experience, I find a careful follow up of these resected cases leaves much to be desired, and perhaps in view of the ease with which transurethral resections can be done with our modern instruments, the technician is liable to be too enthusiastic, emphasizing the procedure rather than the result. Further experience will decide whether the life expectancy of resected patients is equal to, or as long as, that of patients who have had a total prostatectomy.

In traveling during the winter in the South, I have been impressed by the reports of large numbers of "resected" patients who require continued treatment, soundings and bladder irrigations, *etc.*, which practically no properly prostatectomized patient ever requires. There is no doubt that most resected cases void more readily and have much less residual than prior to operation, but large numbers are not absolutely and definitely cured, as they are after a complete enucleation of the adenoma by operation. Pyuria, frequency and dysuria often persist for months or permanently in every series in which a really careful follow up is maintained.

That transurethral resection of the prostate has become an unusually

popular procedure, without due selection of cases, cannot be denied. This is clearly evidenced by the fact that few clinics in the first 30 years of this century were able to report over 1,000 cases in whom prostatectomy had been indicated and performed, whereas in the last five years a number of clinics have reported over 500, and some even 1,000 to 2,000, cases that have been suitable for and required transurethral resection. Is the male in our race degenerating so rapidly, or is every male a potential victim of the resectionist? Even after broadening and extending the indications for treatment of prostatism by prostatic resection, it takes a lot of imagination to believe that this sudden increase in prostatics is not a product of poor, inadequate and slipshod diagnostic investigation. Naturally it follows that in these cases, without the usual imbalance caused by real prostatic adenomata, the mortality of resection must be low, the risk being almost negligible.

We must remember that we are doctors, and not be carried away with technical performances which savor occasionally of tricks. Undoubtedly the popularization of transurethral resection has done infinite damage to patients throughout the country, to mention only the reported great increase in the sale of penis clamps for postoperative incontinence. It may take some time before the fever abates, but from an adequate, carefully studied series of cases, not running into overwhelming thousands, I feel sure that the profession will decide, much as I have done in the above few words, as to the field and scope of transurethral resection.

Dr. William E. Lower (Cleveland, Ohio).—The procedure of transurethral resection of the bladder neck for obstruction has been revived in the last few years and, as Doctor Kretschmer has said, this is probably due to improvements in instruments and electrical apparatus. Not only are the median bars and the sclerotic obstructions resected now, but the use of transurethral resection is being extended to include a much wider field, especially in the treatment of prostatic hypertrophy.

I think one thing Doctor Kretschmer did not stress sufficiently is that transurethral resection is not a simple procedure. Technically, it is more difficult than prostatectomy and the convalescence following this operation is, in many cases, not without complications. If the operation is not well done, incontinence occurs with its associated annoyance.

In what cases should this procedure be used? Experience has taught us that its scope of usefulness is much wider than we formerly thought, but again, I want to emphasize the danger of broadcasting the simplicity of the operation. However, it is true that it carries with it a low mortality rate and, so far, the postoperative results in most cases have been very good. In approximately 500 resections which we have performed, the mortality rate has been 1.8 per cent and we have had a series of 225 cases without a death. These are not selected cases, but include those of all types, as well as many feeble and elderly persons.

I want to emphasize a few things Doctor Kretschmer pointed out. First, the shortening of the preoperative period without catheter drainage. We no longer put in a catheter or make a cystoscopic examination in these patients. If a patient comes in with a history of frequency and especially of nocturia, we plan to make a cystoscopic examination and prepare the patient for a resection. If, at the cystoscopic examination, we find that the obstruction is not suitable for resection, we treat the patient in another way. But if it is suitable for resection, we do it at the same time, thus avoiding catastrophes that sometimes follow the use of the indwelling catheter.

As we have gained wider experience, we have removed larger and larger amounts of tissue. Another advantage of this operation is that a second or

even a third operation may be performed if necessary, without any special risk.

Bleeding is becoming inconsequential. When the procedure was first used, bleeding occurred frequently, but in only one case have we had to open the bladder.

The incidence of epididymitis in the entire series is 6.8 per cent, and the average hospital stay is less than seven days; this economic factor is of considerable importance, especially in these times.

There is no doubt that this method is very practical and of far reaching importance, but its use must be limited to those who have had sufficient experience to perform it properly; otherwise, we are going to have many crippled old men and a high mortality rate from a method which, when handled properly, is an extremely valuable one.

Dr. Hugh Cabot (Rochester, Minn.).—In the first place, there are various methods of whittling out the prostate through the urethra. My notion is that the instruments will probably be greatly modified. But I insist we ought to discuss the problem without prejudice. Doctor Beer has perhaps a case to prove. I think he does chiefly prostatectomy. Doctor Kretschmer has a case to prove. He does chiefly resection. I have no case to prove.

Doctor Beer refers to the unfortunate results of resection. If we would tell the truth, we could load the statistics pretty handsomely with the unfortunate results of prostatectomy. The mortality of prostatectomy, which up to the last few years was in more than half of the cases in the hands of the occasional surgeon, has never the country over been less than 20 per cent, and is not now. Prostatectomy is an operation for experts, whether you use the suprapubic, transurethral, or the perineal method. I insist that we ought to confine our discussion to the results obtained by experts, not the results obtained by people who operate upon the patient not for prostatic obstruction but for a hundred dollars. That will cause damage, and does, in every field of surgery. That is not fair to the operation.

Six years ago, more than half the patients on my surgical list were prostatectomies. I have not done a prostatectomy in three years. There have been only three or four done in the last two years at the Mayo Clinic.

The age group is interesting. One thousand nine hundred twenty-two consecutive prostatectomies, done between 1924 and 1932, showed a 5.6 per cent incidence of patients over 75 years of age. The last 700 cases, up to January 1, 1936, show 15.7 per cent of the patients over 75.

It is frequently suggested that this operation is being performed chiefly for people who do not need it. I do not think that is true. We are operating upon sicker patients and with better results.

It has been the habit at the clinic, and it is not a bad one, to keep all the tissue which the surgeons remove. About a year ago I thought it would be interesting to take a series of 1,000 cases, to give a fair average of the gross amount of prostatic tissue removed by suprapubic prostatectomy, and compare it with the gross amount removed by transurethral resection. The amounts removed by resection are approaching those removed by prostatectomy. The group at the clinic doing resections now take out almost as much tissue on the average as was taken out by prostatectomy five years ago. It thus appears the transurethral method is becoming the method of prostatectomy. If we insist upon the same amount of skill for this method as is shown by the experts using the suprapubic or the perineal route, then resection has much to offer and will cover a large portion of the field.

Dr. Herman L. Kretschmer (Chicago, Ill.) closing.—I agree with

Doctor Beer that a great deal of harm is done by enthusiasts, whether they be in the field of general surgery, urology, or internal medicine. I have tried to approach the problem of transurethral resection with an open mind, without any preconceived notions, and have tried not to become hyperenthusiastic.

Just as the occasional patient may fail to receive symptomatic relief after an appendectomy, so there may be patients who do not obtain symptomatic relief, or are apparently worse, after resection. Close analysis of this group will show that the obstruction has not been completely relieved in some cases; in others, the complaints are due to an overlooked carcinoma or the presence of a diverticulum or, in a rare instance, are due to a large hydronephrosis.

Regarding the number of resections: In my experience it has been necessary in about 10 per cent of the cases to perform a second resection, and, as I mentioned in the paper, the cases in whom a second resection was necessary fall into three groups: first, the cases of very large hypertrophies in whom it is evident that a second resection was necessary to remove the obstruction; second, the cases of carcinoma that recur or that continue to grow and produce obstruction; third, the small number of cases in which not enough tissue had been taken off at the time of resection. There were five patients who required three resections each.

I agree with the statement that has been made that it is unfortunate that there has been so much sales promotion regarding various types of machine and not enough stress has been placed on thorough grounding in pathology and endoscopy of the prostatic urethra and bladder neck.

Regarding the question of incontinence, I would state that we have no patients with complete incontinence; there were two patients who had some slight trouble and they are rapidly improving.

I wish to emphasize what Doctor Lower said, namely, that this is not a simple operation and that technically it is very difficult to carry out.

Regarding the selection of cases, I have tried to emphasize in this paper that this is still a moot point for discussion. An interesting thing was noted in this series of cases, namely, that the patients who had late secondary bleeding, that is, who had bleeding two or more years after resection, were all patients who had very large hypertrophies in the beginning.

I wish to agree with the rest of the points made by Doctor Cabot and Doctor Lower and it seems to me that this procedure will take its proper place in our armamentarium in the very near future.

RELIEF OF PAIN BY PERIPHERAL NERVE BLOCK IN ARTERIAL DISEASES OF THE LOWER EXTREMITIES*

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FORTY-SIX cases of ulceration and gangrene in the lower extremities caused by occlusive arterial diseases (arteriosclerosis with and without diabetes mellitus and thrombo-angiitis obliterans) were treated by the author at the Presbyterian Hospital between 1930 and 1935, by peripheral nerve block in the lower third of the leg.

The operations were performed by exposing the nerves and blocking them with either one or a combination of the following methods: injection of alcohol and crushing, division, division and sutures, or by crushing 2 to 6 Mm. of the nerve.

The foot receives its nerve supply from the posterior tibial which supplies the skin of the sole, tarsal and metatarsal joints, skin of the plantar medial side of the great toe, and adjacent sides of the second, third, fourth and fifth toes. The sural supplies the skin of the heel, lateral side of foot and fifth toe and, occasionally, the lateral side of the fourth toe. The deep peroneal supplies the ankle joint, tarsal joints, metatarsophalangeal joints of the second, third and fourth toes, adjacent sides of the great and second toes, and the first metatarsophalangeal joint. The superficial peroneal supplies the skin over the greater part of the dorsum of the foot, lower part of the leg anteriorly and laterally, medial side of the great toe, adjacent sides of the great and second toes, skin of the medial side of the ankle and foot, contiguous sides of the third and fourth and fourth and fifth toes, lateral side of the foot and ankle, and dorsal surfaces of all toes except the lateral side of the fifth toe. Frequently some of the lateral branches are absent and their function is taken over by the sural.

The internal saphenous nerve supplies the skin of the medial ankle region and medial side of the foot as far as the ball of the great toe.

Verneuil,[20] in 1894, reported a case in which Quenu, in 1893, resected portions of the internal and external popliteal and internal saphenous nerves in the popliteal space because of pain from gangrene of the leg, due to exposure, in a patient 75 years old. On admission his condition of extremis prevented radical amputation. He was entirely relieved of pain by the nerve division. Chauffard described a case, reported by Quenu,[14] in 1910, operated upon by Duval, in 1909, for gangrene of the great toe in which he sectioned the anterior and posterior tibial, internal saphenous and musculocutaneous

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nerves at the ankle for excruciating pain. The patient experienced immediate and complete relief.

Silbert,[17] in 1922, reported three cases of thrombo-angiitis obliterans in whom he exposed the posterior tibial nerve under novocain anesthesia and injected it with absolute alcohol at the internal malleolus and obtained relief from pain in the foot.

The following are various treatments reported for the relief of pain in the lower extremities: ligation of the peripheral arteries and veins[9, 12, 13]; periarterial sympathectomy[2, 10, 16]; periarterial injection of alcohol[7, 11]; dorsal and lumbar sympathectomy ganglionectomy[1, 4]; injection with alcohol about dorsal and lumbar sympathetic ganglia[6, 16]; intravenous injection of foreign protein[3, 4]; intravenous injection of hypertonic (5 per cent) saline[17]; division of subcutaneous nerves about painful ulcerations[5]; pressure-section treatments and peripheral nerve block.[18, 19]

Smithwick and White,[18] in 1930, were the first to report a systematic technic for alcohol injection of the sensory nerves in the leg in order to render the foot insensitive. They advocated exposing the nerves low enough in the leg so that the injection did not paralyze the muscles of the leg and sufficiently proximal to the lesion to anticipate a reasonable chance of primary operative wound healing.

In 1935, the same authors[19] reported 28 cases of thrombo-angiitis obliterans and 17 of arteriosclerosis with extensive obliteration of vessels, severe pain, ulceration and gangrene, in whom conservative treatment had failed and peripheral nerve block by alcohol injection was performed.

Laskey and Silbert,[8] in 1933, recommended nerve section and suture in preference to alcohol injection because it was impossible to be sure that all of the nerve fibers came in contact with the alcohol and alcohol leaking from the nerve into the tissues might cause necrosis.

Peripheral nerve block is an anatomic operation which should not be attempted until the operator has familiarized himself with the exact local anatomy. Poor wound healing may be attributed to extensive dissection because of lack of anatomic knowledge and failure to appreciate the amount of operative trauma these tissues will tolerate. Perfection of operative technic on anatomic specimens is recommended before the operation is attempted on patients.

Procedure.—Infiltration anesthesia with one-half per cent novocain (without adrenalin) is used.

The Posterior Tibial Nerve is exposed through a 5-cm. incision 15 cm. above the internal malleolus and 2 cm. posterior to the medial tibial edge. The incision should be posterior to the internal saphenous vein. The internal saphenous nerve lies just posterior to the vein in the subcutaneous tissue and can be blocked at this time if desired. The deep fascia is incised, the septum between the flexor digitorum longus and the gastrocnemius-soleus group is followed laterally by blunt dissection to the nerve-vessel bundle which is retracted laterally, incised posteriorly and the nerve which lies posterior to the

vessel is isolated. It is freed for 1 cm., injected with 1 cc. of one per cent novocain and crushed once, twice or three times with a small hemostat, depending upon whether it is desired to obtain anesthesia of the part it supplies for six, nine or 12 months. For alcohol injection block the nerve is freed for 2 cm., silk ligatures are placed to completely encircle it, 1 cm. apart, the nerve is drawn out of its bed, saline compresses are placed behind and around it and 1 to 1½ cc. of either absolute or 85 per cent alcohol is injected into the nerve through a 26 gauge needle which is introduced on a slant, for 1 cm., into the nerve. The injected nerve becomes white and distended. The blood in the vessels of the nerve becomes brown. By means of the encircling sutures, which hold the injected alcohol between them, the nerve is rotated and injected posteriorly and medially not using more than 1½ cc. of alcohol for the complete injection. The encircling sutures are removed, the nerve is dropped back into its sheath, hemastasis is effected with five zero plain catgut, the deep fascia is not sutured and the skin is gently approximated with fine silk on sharp, small, cutting edged needles.

Such a block will produce anesthesia in the sole supplied by this nerve for six to 12 months. Crushing the nerve in the injected area will prolong the anesthesia for four to six months more. Division and suture requires an exposure of the nerve for 2 to 4 cm. Sutures of fine silk are placed before the nerve is divided and the vessel of the nerve is ligated before complete division. This type of block is a more complicated operative procedure than crushing which produces the same results without ill effects and unless there is definite indication for division and suture I prefer crushing the nerve. Nerve crushing is the simplest, quickest and least traumatizing to soft parts and is recommended as the method of choice.

The Deep and Superficial Peroneal Nerves may be blocked through the same or separate incisions. The operator should decide whether one or two incisions will be used before operating. In extremities, with severely compromised circulations, separate small incisions placed directly over each nerve are more likely to heal by primary union than a single large incision exposing both nerves, which may require a dangerous amount of retraction and subcutaneous tissue dissection. To expose the deep peroneal nerve through a single incision, the skin is incised for 3 cm., 12 cm. above the external malleolus just lateral to the tendon of the tibialis anticus muscle. To expose the deep and superficial peroneal nerves through a single incision, the skin is incised for 5 to 6 cm., 12 to 15 cm. above the external malleolus. The deep peroneal nerve lies deep to, and between the tendon of, the tibialis anticus and extensor digitorum longus muscle, anterior to the vessels, about 1 cm. below the deep fascia and it is not a large nerve. It may be posterior to the vessels. This nerve should be divided only for the relief of pain in the presence of a lesion which will not heal, and when amputation of the great or second toe is imminent. If the toe lesion is expected to heal, the nerve is blocked by crushing, alcohol injection or both.

The Superficial Peroneal Nerve is larger than the deep peroneal. A single

incision to expose it is made 2 cm. long, 8 to 10 cm. above the external malleolus midway between the tibia and fibula. It is placed sufficiently posterior to the incision exposing the deep peroneal to insure sufficient blood supply to the skin between the incisions. The nerve trunk or its two separate branches at this point should be blocked. Thin extremities are best suited to a single incision exposing both deep and superficial peroneal nerves. The incision is placed 12 to 15 cm. above the external malleolus over the extensor digitorum longus muscle and should be about 6 cm. long.

The Sural Nerve lies in the subcutaneous tissue and is exposed through a 1 cm. incision 8 cm. above the external malleolus just lateral to the tendo achillis. It lies deep to the small saphenous vein.

The toe or foot lesion is not surgically treated at the time of the nerve block. It is dealt with more radically after primary operative wound healing has occurred.

Incision should not be placed near acute pathology in the leg; in fact, such pathology contraindicates nerve block until it has subsided. Occasionally, nerves are not found in their usual anatomic position, in which case it is dangerous to perform extensive dissection looking for them. It is better to close the wound, wait for healing, and then locate the nerve by novocain injection and make the incision over this site. Unusual branching and abnormal distribution of the nerves may be encountered and should be borne in mind when a nerve is being sought for, otherwise complete anesthesia may not be obtained over the usual anatomic distribution of the nerve.

In the 46 cases of obliterative arterial diseases of the lower extremities observed, 82 nerves were blocked with alcohol; 29 with alcohol and crushing; 27 by division and three by division and suture. The following results are reported from a total of 141 blocked nerves. In this series the following is interesting. There was one female with pathologically proven thrombo-angiitis obliterans. The average age of the thrombo-angiitis obliterans group was 39 years; the arteriosclerotics with diabetes mellitus, 62 years, and the arteriosclerotics without diabetes mellitus, 66 years. The dorsalis pedis and posterior tibial arteries were not palpable in any of the cases blocked. The popliteal artery was palpable in 50 per cent of all cases blocked and the femorals were palpable in all cases. The oscillometric readings in the lower third of the leg varied from 0 to 1. The surface temperature of the toes were all two to three degrees below normal but were normal for the proximal half of the foot and lower legs. Pain was entirely relieved in 97 per cent of the thrombo-angiitis obliterans cases, in 90 per cent of the arteriosclerotics without diabetes mellitus, and 81 per cent of the arteriosclerotics with diabetes were relieved of from 75 to 90 per cent of their pain. The wound healing was primary in 91 per cent of all cases. In three of the earlier cases skin necrosis prevented complete healing for two or three months. The follow-up in the thrombo-angiitis obliterans cases has been uniformly good. The return of sensation has been preceded by paresthesia which has not been trouble-

TABLE I

THROMBO-ANGIITIS OBLITERANS

				Arteries				*Nerve Block*							*Amputations*			
Pt.	Unit No.	Age	Sex	DP.	PT.	P.	F.	Osc.	Post. Tib.	Ant. Tib.	Sup. Per.	Sural	Pain Relieved	Wound Healing	Cont. Gang.	Leg	Thigh	Art. Leg	Days—Block Amp.	Follow-Up
F. C.	80319	47	M.	—	—	P	P	0	A	A	A		100%	P	Yes	Gritti-Stokes		Yes	51	4 yrs. 334*
R. T.	288299	41	M.	—	—	P	P	2	A	AC	A	A	100%	P	Toe	Toe				2½ yrs. 444
H. B.	330722	28	F.	—	—	—	P	.5	DS	A	A	D	85%	P	3 toes	3 toes				3 yrs. 444
J. M.	278427	40	M.	—	—	—	P		A	A	A		100%	P	No					4½ yrs. 444
C. H.	361398	40	M.	—	—	—	P	0	A	C	C	A	75%	P						
S. F.	191402	51	M.	—	—	—	P		A	C	A		80%	P	Yes	L1/3		Yes	27 mos.	2 yrs. 444
J. O.	259843	47	M.	—	—	—	P	2–2.5	A	AC	AC		100%	P	Toe	Toe				5 yrs. 444
	(Left)			—	—	P	P		C	C	C	C	100%	P						
J. M.	356049	36	M.	—	—	P	P	.5	A	A	A		100%	P	No					3 yrs. 444
A. R.	366193	36	M.	—	—	—	P	.25	A	D	D		100%	P	Toe	Toe				2 yrs. 444
				—	—	—	P		(Rt) A	A	C		100%	P						
									(Rt) D-S	D			90%	S						
									(Rt) D-S	D	D		100%	S						
D. S.	363519	43	M.	—	—	—	P	2–¼	A	A	A	A	0%	P						Pain in anesthetic area for 6 mos. Psychopathic. Duod. ulcer
J. W.	359197	47	M.	—	—	P	P		A	A	A		100%	S	No	Lat. wd. ulcerated 5 mos. after nerve block. Culture: Pyocyaneus (in pure culture).				
L. R.	249991	41	M.	—	—	P	P		A				50%	P	No					
A. H.	392546	25	M.	—	—	P	P	0	A	AC	A	AC	100%	P	No					2 yrs. 444 phlebitis cont.
G. H.	443751	35	M.	—	—	P	P	0	AC	AC	AC	AC	100%	P	Yes	L1/3		Yes	7 wks.	Ulcer on amp. stump
C. J.	44502	38	M.	—	—	P	P	0	A	A	A	A	100%	P	No					7 mos. 444
S. S.	410276	52	M.	—	—	P	P		C				100%							
F. M.	244746	28	M.	—	—	P	P		A	C	C		100%	P	Toe	Toe	Typhoid vaccine failed to relieve pain			3 yrs. 444
									(Lt)	A	A		100%	P	2 toes					2 yrs. 444
									(Lt) A				100%	P			Thumb and finger amp.			
									(Lt)			A		P			Brachial periarterial sympathectomy failed to relieve pain			

A—Alcohol injection, AC—Alcohol injection and crushing, D—Division, DS—Division and suture.

* Follow-Up, Anatomic—(A), Symptomatic (S), Economic (E), four equals 100 per cent, three equals 75 per cent, two equals 50 per cent, one equals 25 per cent of normal, so 4444 equals 100 per cent, Anatomic, Symptomatic, and Economic Follow-Up results.

TABLE II

ARTERIOSCLEROSIS

				Arteries					*Nerve Block*							*Amputations*				
Pt.	Unit No.	Age	Sex	DP.	PT.	P.	F.	Osc.	Post. Tib.	Ant. Tib.	Sup. Per.	Sural	Pain Relieved	Wound Healing	Cont. Gang.	Leg	Thigh	Art. Leg	Days—Block Amp.	Follow-Up
A. O.	230891	56	M.	—	—	P	P		A	C	A		60%	PP	No	No	No	No		Two yrs. 322.*
										D	D	D		Second						Died after prostatectomy
L. B.	406170	62	M.	—	—	—	P	¼	A-C	D	D	D	90%	PP	Yes	L1/3		Yes	70	18 mos. 444
C. W.	356223	66	M.	—	—	—	P	¼	A	A-C	A		85%	PP	No					2 yrs., 10 mos.
J. T.	392510	69	M.	—	—	P	P	¾	A	C	C	A-C	100%	PP	Yes	L1/3		Yes	11	2 yrs. 444
E. O.	307215	72	M.	—	—	—	P		A	A	A	A	Amp. through thigh 2 days after nerve block and amp. 5th toe. Died 3 days later C. Welch. P.							
S. R.	411423	68	M.	—	—	—	P	0	A	A	A		90%	Slow	Yes	L1/3		No	40	Fractured neck of femur same side
L. K.	141655	58	M.	—	—	P	P		A	D	A		100%	PP	Yes	L1/3		Yes	150	3 yrs. 330
J. P.	369121	82	M.	—	—	P	P	0	A	D	D	A-C	100%	PP	No					Cardiac death 2½ yrs. after block
E. E.	384822	45	M.	—	—	—	P	0	A	D	A-C	A	100%	PP	Yes	5 toes				2 yrs. 244 2 yrs. 030 Ulcer dorsum persists
J. O.	435999	64	M.	—	—	P	P	0	A-C	D	C		90%	PP	Yes	L1/3			38	Nerve anomaly (AT)
S. L.	300503	52	F.	—	P	P	P		A	A	A		90%	PP	No					
T. T.	441848	67	M.	—	—	P	P	1.5	A-C	D	A-C		90%	PP	Yes	L1/3		Yes	30	8 mos. 444
B. W.	436003	75	F.	—	—	—	P	0	A-C	D	A-D	A-D	90%	PP	Yes	L1/3		No	14	Died 2 mos. after amp. bronchopneu.
M. H.		71	M.	—	—	—	P	0	A-C	A-C	A-C	A-C	80%	Slow	Yes	No				4 mos. 030
J. K.	461345	79	M.	—	—	P	P	0	A-C	D	A-C		100%	PP	Yes	No				
J. S.		60	M.	—	—	P	P	0	A-C	D	D		95%	PP		Gt. toe				8 mos. 444
O. F.	470134	70	M.	—	—	—	P	0	C	C	C	C	100%	PP						
F. R.	448400	68	F.	—	—	—	P	0	C	C	C	C	100%							

A—Alcohol injection, AC—Alcohol injection and crushing, D—Division, S—Suture after division.

Arteries (—) not palpable, (P) Palpable.

Nerves (A) Alcohol Injection, (C) Crush, (D) Division.

* Follow-Up, Anatomic—(A), Symptomatic (S), Economic (E), four equals 100 per cent, three equals 75 per cent, two equals 50 per cent, one equals 25 per cent of normal, so 4444 equals 100 per cent, Anatomic, Symptomatic, and Economic Follow-Up results.

TABLE III

DIABETES MELLITUS AND ARTERIOSCLEROSIS

				Arteries					*Nerve Block*						*Amputations*				
Pt.	Unit No.	Age	Sex	DP.	PT.	P.	F.	Osc.	Post. Tib.	Ant. Tib.	Sup. Per.	Sural	Pain Relieved	Wound Healing	Cont. Gang.	Leg	Thigh	Art. Leg	Days—Block Amp.	Follow-Up
H. P	369316	59	M.	—	—	—	P	o	AC	D	D	D	90%	S	Yes	L1/3		Yes	25 days	2½ yrs. 444*
D. I.	283294	69	F.	—	—	—	P	o	A	D	D	D	90%	P	Yes	L1/3		Yes	34 days	2 yrs. 444. Other leg amp. 2 yrs. later patient died after this amp.
A. C.	261072	63	M.	—	—	—	P		A	C	A		100%	P(2wd) S(1wd)	Yes	M1/3		Yes	32 days	5 yrs. 444. Amp. followed heat burn of external heat after block
C. B.	250358	74	F.	—	—	P	P		A	A	A		60%	P	Yes	U1/3		Yes	25 days	5 yrs. 444
M. K.	76823	56	F.	P	P	P	P		A	A	C		100%	P	No					1½ yrs. 444. Died 1½ yrs. after block of B. Friedlander pneumonia
A. R.	352879	49	M.	—	—	—	P		A Ligation femoral vein A D	A	A	A	100% 40% 60%	P S P	No Yes Yes		Gritti-Stokes	Yes	1 yr.	1 yr. 444 2 yrs. 330. Burned heel with external heat after block. Burned toes
E. W.	239180	70	F.	—	—	—	P	o	AC	AC-D	D	D	90%	P	Yes	L1/3	L1/3	No	30 days	Burns did not heal Second leg amp. through L1/3 thigh. Recovered. Pt. confined to bed and chair
M. F.	441848	61	F.	—	—	P	P	o	AC	AC	AC	AC	100%	P	Yes	L1/3		No	48 days	Amp. wd. left open, healed by granulation. 4 mos. 220
M. W.	446317	67	F.	—	—	P	P	1	AC			AC	50%	P	Yes	L1/3		Too soon	120 days	3 mos. 340
E. R.	312478	71	M.						A	AC	C	C	75%	P	No	Toe			60 days	1 yr. 444
L. L.	252371	43	M.	—	—	—	P		A	A	A		100%	P	Yes		M1/3 (At another hospital)			1 yr. after block. 5 mos. 440

A—Alcohol injection, AC—Alcohol injection and crushing, D—Division.

* Follow-Up, Anatomic—(A), Symptomatic (S), Economic (E), four equals 100 per cent, three equals 75 per cent, two equals 50 per cent, one equals 25 per cent of normal, so 4444 equals 100 per cent, Anatomic, Symptomatic, and Economic Follow-Up results.

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some. The foot has remained somewhat warmer and the local lesions have healed or else toe amputation sites in the anesthetized foot have healed. In three cases blocks of the same nerves have been performed three times for a recurrent painful toe ulceration over a five year period. In the arteriosclerotic cases, without diabetes mellitus, healing of the toe lesion did not occur in 50 per cent of the cases and the block was followed in two to four weeks by amputation through the junction of the middle and lower third of the leg by a modified guillotine technic.[21] The results in the arteriosclerotic group with diabetes mellitus was the poorest of either group and more amputations followed in these cases.

TABLE IV

AMPUTATIONS IN 46 CASES OF PERIPHERAL NERVE BLOCK

	No. Cases	Leg	Thigh	Gritti-Stokes	Toes	No Amputation	Number Wounds Healing by Primary Union	Number Wounds Healing by Secondary Union
Thrombo-angiitis Obliterans	17	2	0	1	5	9	51	3
Arterio-sclerosis without Diabetes	18	7	1	0	3	8	52	3
Arterio-sclerosis with Diabetes	11	8	2	1	1	1	31	3
Totals	46	17	3	2	9	18	134	9

22 of 46 cases or 48% had a major amputation.
9 of 46 cases or 19% had a toe amputation.
18 of 46 cases or 39% had no amputation.

In the thrombo-angiitis group there were three major amputations, two through the lower leg and one Gritti-Stokes.

In the arteriosclerotic group without diabetes there were eight major amputations, seven were through the lower leg and one through the thigh.

In the arteriosclerotic group with diabetes there were ten major amputations of which eight were through the lower leg and two were through the thigh.

CONCLUSIONS

The chief indication for peripheral nerve block is to relieve pain. It is a palliative operation. It saved 50 per cent of the arteriosclerosis cases and 20 per cent of the arteriosclerotics with diabetes from a major amputation. No leg amputations were performed in the thrombo-angiitis group. The release of vasoconstriction by block increased the local temperature in the anesthetized area to varying degrees in the thrombo-angiitis cases, depending upon the degree of arterial occlusion and spasm in the vessel wall. It made dressings, treatments, postural exercises, warm whirlpool baths, dakinization,

excision of sloughs and osteomyelitic sequestra and drainage of local abscesses, possible without subjecting the patient to multiple general anesthesias.

Careful instruction is necessary concerning foot hygiene and properly fitting shoes with corrected weight bearing to prevent trophic ulceration in the insensitive member. Patients should be particularly warned not to expose the insensitive part to any form of heat. Nerve block has permitted recovery with fewer major amputations and made it possible to perform minor ones where major ones might primarily have been indicated. The relief of pain during wound healing has freed patients from sedatives, improved their appetites, increased their weight and permitted normal function of their gastro-intestinal and genito-urinary tracts which were interfered with by sedatives. Restful sleep has improved cerebral disorientation. Paralysis of the intrinsic muscles of the foot which follows any type of nerve block has not interfered with normal locomotion.

These observations are in accord with many of those of previous essayists. For restatements I am apologetic but they are made to emphasize important points of technic and treatment and serve to verify other previously reported experiences.

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DISCUSSION.—DR. GRANT P. PENNOYER (New York) stated that in the vascular clinic at Roosevelt Hospital experience with the procedure described by Doctor Smith had been sufficient to be convincing that in a certain very limited number of cases the procedure is extremely valuable. Before it is attempted, however, the indications must be considered very carefully. The case should be one suffering very severe pain from a foot lesion requiring conservative treatment over a long period of time. Quite a few patients with peripheral vascular disease fall in this group. Opiates with their depressing effect on the aged are avoided. The freedom from pain greatly improves the general condition of the patient. Minor operations such as a separation of sloughs and incisions for drainage can be performed without any anesthesia. The operation is not technically difficult and the results are very satisfactory.

Doctor Pennoyer cited five cases in which peripheral nerve sections had been performed for the relief of pain in the foot, which, he admitted, was a very limited series upon which to base any conclusions. In one case the operation was only a partial success, the patient's pain not being entirely relieved. The reason for this might have been some anatomic anomaly of the peripheral nerves or a technical error in identifying them. In the other four cases, however, the relief of pain was complete. Two of these patients had arteriosclerotic gangrene which slowly progressed so that they later required amputation, but they both had a considerable period, one of about six weeks and the other four, before amputation was performed, during which they were quite comfortable. Before the nerve section they both had so much pain that it could be only partially controlled by morphine. The other two cases both have their feet, except for the loss of toes. One of these was an old gentleman with an arteriosclerotic gangrene of his right great toe who later developed an osteomyelitis of the first metatarsal. It seemed as though he was going to lose his foot but he was made entirely comfortable by blocking the nerves, and with conservative treatment for four months lost only his great toe and the distal half of the first metatarsal.

An interesting anatomic sidelight in these cases is the fact that formerly

it was taught in the Anatomy Department of Columbia University that the intrinsic muscles of the sole of the foot are very important in balancing when standing on one foot. It has now been noted that despite the fact that these muscles are necessarily completely paralyzed when the posterior tibial nerve is divided, these people seem to be able to walk and balance themselves on one foot about as well as they could before.

At Roosevelt Hospital they acutally divided the nerves instead of crushing them or injecting them with alcohol.

DR. DAVID C. BULL (New York) heartily endorsed Doctor Smith's description of the results of this procedure, namely, relief of pain, decrease in morphine required, *etc.* He called attention to a complication which might be overlooked in thrombo-angiitis obliterans, but which had occurred in one of his cases. The small wound that had been made for the peroneal nerve approach healed primarily within a week. The patient went home soon afterward, only to return several weeks later with a spreading ulcer originating in the wound. On culture this yielded a pure growth of *Bacillus emphysematosis.* The lesion cleared up with wet dressings and skin graft, and healed completely.

NERVE INJURY IN FRACTURE OF THE PELVIS

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DURING a recent one year period, 18 patients with fracture of the pelvis were treated in the Henry Ford Hospital. Six of these presented definite evidence of nerve injury. While the complaints and symptoms were not serious, in terms of life and death or ultimate function, the nerve damage caused such severe neuralgia in four of these cases that the neurologic complication became the chief complaint throughout the period of hospitalization. This experience prompted our study of 100 consecutive cases of fracture of the pelvis with special reference to nerve injury, and a review of the literature on the subject.

The standard text-books mention the complication briefly or not at all. Scudder[24] states that G. G. Davis observed nerve injury in 2 per cent of pelvic fractures, and that the trauma to the nerve is in the origin of the lumbosacral cord as it lies at the pelvic brim. It is also stated that the evidence of nerve damage appears immediately, and that the likelihood of recovery from peroneal palsy incurred in this way is not great. Wilson and Cochrane[27] do not mention the complication. Speed[25] says, "Nerve injury to the sacral plexus is rare, unless there is a severe crushing injury of the sacrum." Under the caption "Rare Complications of Fractures of the Pelvis," Key and Conwell[23] state that paralysis of the sciatic nerve occasionally occurs in fractures of the posterior pelvic ring. The paralysis is usually temporary and clears up spontaneously. Paralysis of the obturator nerve is a rare complication of fractures of the anterior pelvic ring.

A review of the English and German literature of the past 20 years revealed no paper dealing particularly with this complication. No attempt was made to review the entire literature on fractures of the pelvis, a considerable part of which deals especially with urinary tract injury, and methods of treatment of the fracture. However, a fair idea of the frequency of nerve injury was obtained by reading all the articles containing reports of series of cases, and others in which nerve injury might have been mentioned. One thousand eight hundred eighty-nine cases of fracture of the pelvis were collected, and in this group, 14 cases of nerve damage were mentioned, giving an incidence of 0.75 per cent for the complication. The sources of this material are given chronologically in the bibliographic table appended.

Most of these writers make no mention of nerve injury. Caldwell[2] stated that trauma to the various nerves in the vicinity of the pelvis may occur, but he reported no cases. Bacon had one case of persistent sciatica in a series of 32 cases of fracture. This patient had a fracture of the left ischium with moderate displacement. The nerve symptoms were late in appearing, and

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prolonged the disability to six months. Hirsch[22] observed nerve irritation in his series of 102 cases of pelvic fracture, but the incidence was not stated. He was of the opinion that injuries to the nerve plexus, as demonstrated by disturbance of reflexes, motor function, and sensation, occur most often in injury of the posterior ring of the pelvis. Usually the injury is a spontaneously reparable one, consisting of a tearing, contusion, or hemorrhage into the nerve, and the symptoms subside quickly, with reflexes frequently remaining absent permanently. He did not observe peroneal paralysis.

Among Orator's[5] 61 cases, there were three cases with nerve damage. One case, with fracture involving both sacro-iliac joints, had neuritis and knee contracture which cleared up a year later. Another patient, with a fracture through the left sacro-iliac joint, had paralysis and sensory changes in the extremity 16 years later, and was lame and bed ridden. A third case had a fracture of the left side of the sacrum and complained of sciatica. Gunzer[7] observed two cases of complete sciatic paralysis in his 24 cases. One of these had a Malgaigne fracture, and the other an acetabular fracture. Westerborn[8] noted permanent peroneal paralysis in one case of his series of 306 cases of pelvic fracture, a vertical fracture of the sacrum being present. Sever[9] had one case of fracture into the right sacro-iliac joint, with complete paralysis, sensory and motor, of the right leg. A year later, the patient was well, except for slight weakness of the toes.

G. E. Wilson[28] reported that injury to the sacral plexus is a rare complication of fracture of the pelvis. He had seen only one case. The damage to the pelvis was severe, the right pubic bone having been driven downward two inches, with one-half inch displacement of the ilium on the sacrum on the left side. There was complete left peroneal paralysis, and partial paralysis on the right. There were no sensory disturbances, and the end-result was not stated. In their series, Colp and Findlay[12] observed traumatic neuritis in one case, with fracture of the acetabulum. They assumed that there had been a laceration of the sciatic nerve, and attributed a poor end-result to the nerve injury, with the resultant atrophy of muscles, weakness, and continued pain.

Three cases of peroneal nerve injury were noted by Noland and Conwell[13] in 125 cases. One of these showed permanent disability. Peabody[14] studied eight cases of innominate luxation, and observed sciatica in only one, which case also had a fracture of the transverse process of the fifth lumbar vertebra, in addition to the sacro-iliac dislocation. Wheeler[26] stated, "It is obvious that the lumbosacral cord may be involved in any fracture in the region of the sacro-iliac joint, or the sciatic nerve may be stretched or crushed, giving rise to prolonged and protracted neuralgia." He did not report specific cases.

From a consideration of the above reports, one would conclude that nerve injury is comparatively rare and is to be encountered only in cases with demonstrable injury in the region of the sacro-iliac joint. Injury has been reported only in the sciatic nerve or its peroneal component. The following

nine cases of nerve injury found in 100 pelvic fractures are at variance with the above, because six of them did not present roentgenologic evidence of injury to the posterior ring, although the probability of temporary sacro-iliac separation with spontaneous reduction is admitted. Furthermore, nerves other than the sciatic were involved.

The 100 cases of fracture of the pelvis reviewed in this study were treated during the past ten years. The mortality was 12 per cent, as compared with a mortality of 8.8 per cent in 1,142 cases collected from the literature. There were five ruptures of the bladder and four lacerations of the urethra, or an incidence of urinary tract damage of 9 per cent, as compared with 11 per cent in 1,468 collected cases. The figures on nerve damage, however, differ widely from those obtained from the 1,889 cases reviewed in the literature, the complication occurring in 9 per cent of this series and only 0.75 per cent in the collected cases.

PROTOCOLS OF NINE CASES WITH NERVE INJURY

Case 1.—No. 52539. White male, age 26. Admitted October 24, 1930, after an industrial accident. Roentgenologic examination of the pelvis showed fractures through the superior and inferior rami of the left pubis, and a fracture into the left sacro-iliac joint, with slight separation. There was no evidence of nerve damage on admission. On January 27, 1931, weakness of flexion of the great toe was noted. On February 24, 1931, the patient complained of pain along the course of the sciatic nerve. Sensation was normal. On May 28, 1931, neurologic examination by Dr. G. O. Grain showed slight weakness of the tibialis anticus muscle, the calf muscles, the flexors of the toes, and the peronei. There was absolute anesthesia of the dorsum of the foot and the anterior surface of the leg about two inches above and below the ankle joint. His impression was that this represented residuals of injury to the left lumbosacral plexus. Neurologic examination on August 9, 1931, revealed slight improvement. One year later, the patient made an attempt to go back to work, but was unable to continue. On March 20, 1933, his permanent disability was estimated to be 20 per cent. A year later, he was still complaining of pain in the left foot, and some residual muscular weakness. There was no further follow-up.

Case 2.—No. 177623. White male, age 27. Admitted June 20, 1932, after an industrial accident. Roentgenologic examination of the pelvis showed a fracture of the left side of the sacrum, two fractures of the left ascending ramus of the pubis, and a fracture of the left descending ramus, with considerable separation. He was placed in a pelvic sling, and remained in the hospital 16 weeks. On September 28, 1932, he attempted to walk, but complained of weakness in the left hip and numbness of the left buttock. Neurologic examination showed an area of absolute anesthesia on the left buttock, with muscular weakness not definite at that time. Examination several days later revealed marked weakness of the gluteus medius and slight weakness of the tensor fascia femoris and gluteus minimus on the left. This was assumed to be due to injury to the left sacral plexus, involving the components of the superior gluteal nerve and the cutaneous branches of the first three sacral roots. A hip spica was applied to maintain the hip in abduction. Improvement of the gluteus medius weakness was noted on February 21, 1933, and again on June 16, 1933. On November 24, 1933, it was thought that there had been 100 per cent recovery from the nerve damage, and the patient was working. However, he reappeared February 22, 1936, complaining of some weakness of the left thigh. Slight atrophy was noted, and it was thought that there were some residuals of nerve damage.

Case 3.—No. 153883. White female, age 46. Admitted September 22, 1930, after

an automobile accident, having been treated for several days elsewhere. Roentgenologic examination of the pelvis showed fracture of the superior and inferior rami of the left pubis. An important finding on admission was a severe third degree hot water bottle burn on the lateral aspect of the right thigh, which was infected. The patient stated that the area was numb following the accident, which accounted for her not feeling the overheated bottle. The anesthetized area was in the distribution of the lateral cutaneous femoral nerve. She died suddenly five days after admission, with cyanosis and symptoms of respiratory embarrassment; clinically the cause of death was pulmonary embolism.

Case 4.—No. 211733. White female, age 39. Admitted February 2, 1935, following an automobile accident. There were fractures of the superior and inferior rami of the right pubis, with slight displacement. The patient was treated on a Bradford frame. There were no particular complaints until April 16, 1935, when she was unable to sleep on account of shooting pains in both legs. The pain arose from the low back region and radiated down the posterior aspects of the thighs and legs. Neurologic examination by Doctor Grain two days later revealed absolute anesthesia in the distribution of both lateral cutaneous femoral nerves. That night, the patient filled a hot water bottle herself, and applied it to the right thigh, receiving a third degree burn, slightly smaller than the area of the bottle. After several attempts with Reverdin grafts, the skin defect was closed. The neurologic lesion indirectly prolonged the disability several months.

Case 5.—No. 218804. White female, age 23. Admitted June 22, 1935, following a motor car accident. There were fractures of both rami of the pubis bilaterally, and evidence of bladder damage. She was put on a frame, and suprapubic cystotomy was performed for the ruptured bladder. Four weeks after admission, she complained of pain in the right leg. Neurologic examination showed slight weakness of the extensors of the toes and tenderness of the peroneal and sciatic nerves. The Achilles tendon and the sole of the foot were tender. The knee jerk and ankle jerk was diminished. The symptoms were thought to be due to residuals of mild traumatic sciatic neuritis. There were no further complaints, and the patient was discharged after eight weeks, the suprapubic fistula being closed.

Case 6.—No. 224161. White female, age 28. Admitted October 6, 1935, after an automobile accident. There was a fracture of the superior ramus of the right pubis, without displacement. She was placed on a frame. Preliminary neurologic examination was negative. Two days later, she complained of numbness of the lateral aspect of the right leg. No motor weakness could be made out. On October 23, 1935, she complained bitterly of pain in the leg. Neurologic examination showed no anesthesia of the skin. Slight weakness of the tibialis anticus, tibialis posticus, the peronei, and extensors of the toes was noted; obviously there was partial peroneal paralysis. During the rest of the period of hospitalization, she required constant sedation for the burning pain along the distribution of the peroneal nerve. She was discharged after six weeks, and returned for one follow-up visit a week later, still complaining of a burning sensation in the first and second toes. She was not followed further.

Case 7.—No. 205402. White male, age 39. Admitted January 8, 1936, several days after an automobile accident. There were fractures of the rami of the pubis on both sides, with slight displacement. Bradford frame treatment was instituted. No nerve injury was noted on admission. One day later, it was noted that there were vague sensory changes in the left lower extremity. Neurologic examination the following day showed weakness of the left tibialis anticus muscle, with foot drop. Sensory disturbance was not marked, there being only relative diminution of sensation for touch, pain, and temperature. This represented involvement of the peroneal nerve. The foot drop was still present on January 24, 1936. The patient was discharged in six weeks with no complaints, and on the first follow-up visit a week later, the peroneal palsy had entirely cleared up.

Case 8.—No. 228650. White female, age 22. Admitted January 10, 1936, after an automobile accident. Roentenologic examination of the pelvis revealed a fracture of the body of the right pubis, and the inferior ramus of the left pubis, with negligible dis-

placement. She was placed on a Bradford frame. Ten days after admission, she complained of numbness over the posterior aspect of the left thigh, calf, sole, and dorsum of the left foot. There was no definite anesthesia or motor weakness. The left ankle jerk was weak. On February 5, 1936, she complained bitterly of burning of the sole of the foot, and was frequently in tears from the discomfort, and a neurosurgical consultant suggested subarachnoid injection of alcohol for palliation. The patient refused this procedure, but continued to require large amounts of sedatives. She was discharged after six weeks, still complaining of the burning sensation across the sole of the foot. She did not return for follow-up.

Case 9.—No. 236677. White female, age 26. Admitted February 18, 1936, following an automobile accident. There were fractures of the rami of the pubis on both sides, fracture of the left ilium into the sacro-iliac joint, with considerable overriding of the anterior fragments. She was put on a Bradford frame with ten pounds of lateral traction applied to each hip, accomplished by the use of bilateral leg encasements tied together at the feet, and the traction attached to slings around each thigh. The contraction of the pelvis was satisfactorily corrected, but the plaster encasements were removed early on account of the complaint of pain in the right leg. On March 6, 1936, an area of almost total anesthesia was noted on the anterolateral aspect of the middle of the right thigh. Subsequently, she complained of a burning pain in the ball of the right foot. Diagnosis of traumatic peripheral neuritis was made, the lateral cutaneous femoral nerve being chiefly involved. On March 21, 1936, neurologic check showed a patch of anesthesia on the dorsum of the foot corresponding to the distribution of the superficial peroneal nerve. There was slight weakness of the peroneal muscles. The patient was allowed to be up in eight weeks, and when last seen, was walking with only a slight limp. Sensation was returning to the anesthetic areas, there was no peroneal weakness, and no complaint of neuralgia. It should be noted that the nerve damage was on the right, while the sacro-iliac injury was on the left.

SUMMARY.—The nerves involved in the nine cases cited consisted of injury to the peroneal component of the sciatic nerve in six instances, the lateral cutaneous femoral three times (bilateral in one case), the superior gluteal once, and the posterior branches of the first three sacral nerves once.

The treatment of the nerve symptoms with regard to sensory disturbance was purely symptomatic, and in several cases was unsatisfactory. Several patients appreciated heat in the form of baking or diathermy; others declared that heat aggravated the symptoms, and preferred a cradle over the leg so that the bed clothes did not touch the skin. Subarachnoid injection of alcohol was probably indicated in at least one case, but was not carried out. The lesson to be learned from the two patients with severe hot water bottle burns is evident. The muscle palsies were treated by the usual methods of support until regeneration had occurred.

Since no autopsies or operative procedures were performed upon these cases, there is nothing to add to the theories of pathogenesis of the nerve injuries. The preponderance of sensory over motor disturbance, the tardy appearance of the symptoms, and the spontaneous regeneration, would support the idea of previous writers, namely, that the trauma is in the form of a stretching or contusion, rather than a laceration.

CONCLUSIONS

(1) Nerve injury in fracture of the pelvis is probably more common than is generally recognized.

(2) Such injury may occur in the absence of demonstrable damage to the posterior pelvic ring.

(3) Nerves emerging from the pelvis anteriorly, such as the lateral cutaneous femoral, may be involved, as well as the sciatic.

(4) The protracted neuralgia and muscle weakness may be the chief concern of both the patient and the surgeon in certain pelvic fractures.

The author is indebted to Dr. C. L. Mitchell for permission to publish the case reports.

BIBLIOGRAPHIC TABLE SHOWING INCIDENCE OF NERVE INJURY IN REPORTED SERIES OF CASES

	Total Cases	Nerve Injury
[1] Quain, E. P.: Rupture of Bladder Associated with Fracture of the Pelvis. Surg., Gynec., and Obst., **23**, 55, 1916	127	0
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[12] Colp, R., and Findlay, R. T.: Fractures of the Pelvis. Surg., Gynec. and Obst., **44**, 847, 1929	35	0
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Totals	1,889	14

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THE INFLUENCE OF HEREDITY IN CANCER*

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FROM earliest times there must have been thought and speculation in regard to the effects of heredity on human life. Biblical history states that all Israel was reckoned by genealogies.[1] Heredity plays an important rôle in the size of individuals, their physical shape or habitus, muscular strength, color of the hair and eyes and in many other characteristics. It is only a short step farther to the realization that a definite tendency to disease is also transmitted in certain families. Hemophilia, diabetes and cardiovascular disease illustrate this and there are many others.[2, 3]

The question of an hereditary tendency in cancer patients has evoked much discussion. According to Kurbler,[4] Paget (1857) found such a taint in 22 per cent of 254 cancer patients, but Winiwarter (1878), in 548 cancer patients in Billroth's clinic, found it in only 1 per cent. Siegrist gives 2.5 per cent and Baker (1866) as high as 43 per cent. Most of these observations were from the figures of insurance companies and obviously the conclusions are not to be considered reliable. The differences are too great. As recently as 1908 Bashford[5] made a statistical analysis of the records of the Imperial Cancer Research Fund and concluded that cancer is probably always acquired. Until our records for many generations are more perfect than at present, the statistical approach to this problem will be suggestive only.

In an analysis of family histories at the Eugenics Record office of the Carnegie Institution of Washington, Little[6] shows that the progeny of a cancerous mother and noncancerous father were found to contain an excess of cancer. His study "seemed to show beyond any question that heredity plays a rôle in determining cancer incidence in man."

Wells[7] states that it seems safe to maintain that the existence of an hereditary influence on the susceptibility and resistance to cancer has been established for both man and animals. Maud Slye[8] also believes this to be the fact and suggests that cancer susceptibility behaves like a recessive, insusceptibility like a dominant characteristic. Macklin,[9] using a different statistical approach, very logically shows that the family in which a mother and three daughters had their breasts removed for cancers before the age of 21 years had a 100 per cent incidence of the cancer, while the figures for 1928 in Canada for cancer of the breast at that age show the normal incidence would be 1 in 1,090,000. She feels that there are numerous families in which the incidence of cancer is so high that it precludes any explanation that it is by chance alone that these persons were affected. History and medical literature are full of strikingly similar instances.

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Cancer is said to have been frequent in the family of Napoleon. Napoleon himself, his father, his brother and two sisters were all victims of cancer. Warthin's family S shows 38 members dying of cancer through four generations. Finney[10] reports a family in which the mother, four daughters and three nieces have all been operated upon for cancer. All but one had cancer of the breast. Three of the daughters had a second cancer in the other breast, and one of these had a third cancer in the stomach. There are, however, 37 other members of the present adult generation of sisters, brothers and cousins who are free from cancer. The proportion of diseased individuals and the location of the cancer seem to remove the element of chance. Kurbler[4] describes a cancer family. In the second generation, one woman and two men died of cancer of the stomach. One woman also died of uterine and one woman of hepatic cancer. A son of the woman who had gastric cancer died of cancer of the rectum and all three daughters of the cancer of the liver case had cancer of the breast. In the fourth generation, there were also two women with cancer of the breast. The author recently removed the breast of a woman 38 years old. Her sister had had a radical mastectomy at the age of 36, followed a year later by removal of the other breast, and died of cancer six months later, at the age of 38. Pack[11] cites two brothers age 52 and 54 who were both in the hospital at the same time with adenocarcinoma of the stomach. One came in for tumor and pain, while the other showed loss of weight only. A roentgenologic study demonstrated the growth in both cases. Very convincing evidence is submitted by McFarland and Meade,[12] who feel that the simultaneous occurrence of similar, symmetrical tumors in both of homologous twins tends to prove the genetic origin of such tumors. They have collected a series of 40 tumors occurring symmetrically and simultaneously in 20 pairs of monozygotic twins. These were equally divided between benign and malignant growths. These authors[13] describe carcinomata of the stomach occurring simultaneously in uniovular twins, who were 70 years old. Their symptoms were identical, and the tumors were symmetrically located in the stomach. There was rapid progression of the disease and a similar pathologic report. Both tumors were adenocarcinomata of the cardia and fundus which extended nearly to the pylorus. The symptoms started in October, 1933, in one, and in November, 1933, in the other. One died on April 29, 1934, and the other on June 17, 1934. Croon[14] described adenocarcinomata of the uterus in twins who were both 50 years of age.

Deelman,[15] after an investigation of the family history of 350 cases of cancer and 250 control cases, concludes:

(1) "In a series of families of cancer patients, cancer is more frequent than in any other series of families.

(2) "The surplus of cancer in the family of cancer patients always accumulates in certain distinct families.

(3) "There is a large group of cancer patients in whose family cancer

absolutely is not more frequent than agrees with the 'normal' chance of dying from cancer.

(4) "Hereditary influences are only evident in a small category of cancer cases."

Hunter,[16] using more modern statistics from insurance company records, draws the same conclusions as Deelman but states them differently. He says, "There are undoubtedly families which show a strong predisposition to the disease (cancer), but these are rare in proportion to the total population. So far there has been no conclusive proof that a person with one death in a family, either a parent or brother or sister, need fear a predisposition to the disease." It has been generally accepted that mice transmit all the Mendelian factors necessary for susceptibility to transplanted tumors and also that hereditary factors determine the characteristics of both the host and the tumor cell.[17] Macklin[18] has summarized the subject very well and concludes in part that:

(1) "Cancer is due to an inherited factor.

(2) "Cancer of a specific type, in a specific organ, at a specific age, tends to occur in families.

(3) "Related persons were affected ten times as often as unrelated persons.

(4) "Chronic irritation, when present, appears to accentuate the speed of a reaction which was destined to occur at a later date.

(5) "Identical twins, who have the same type of tumor, in the same organ and frequently at the same age, afford excellent proof of the rôle which inheritance plays in the production of cancer.

(6) "Belief in the inheritance of cancer affords an opportunity for early diagnosis."

She analyzed the life history of a family, later described, in which the mother, three daughters and a son all died of carcinoma of the rectum. Using the 1931 vital statistics for Canada, she found "123 deaths from rectal cancer between the ages of 30 and 55, the ages in which your group died. There were 104,517 deaths that year from all causes, at all ages. Therefore, approximately one in every 750 persons was dying from rectal cancer at those ages. Now, if cancer were due to chance, the chances for rectal cancer affecting five persons in the same family between the ages of 30 and 55 would be determined by the binomial theorem, as follows: (a plus 750b).[5] If that is expanded, you find that in 237,304,687,500,000 families of five persons, you will get one family where all died of rectal cancer without there being any necessity for heredity being brought in as the explanation. If heredity is the explanation, and if it were due to a dominant factor, then, with a mother showing it and a family of four children, you would find the chances for all four affected varying from one in one, if the mother were homozygous for rectal cancer, to one in 16, if she were heterozygous. The chances would be also one in 16 for all children affected, if cancer were due to a recessive, for in this family, the mother would show it and the father would be hetero-

zygous for it, and the results would be the same as if it were due to a dominant with the mother having one factor for it, in other words, heterozygous." Maud Slye was recently quoted as having stated that, in her opinion, cancer could be conquered only by breeding it out of the race. She estimated it would take 500 years to accomplish this.

Abridged Histories of a Family Personally Observed

Case Reports.—The mother, Sophie R., in 1916, at the age of 54 complained of frequent, small, lumpy stools and a sensation of pressure in the rectum and back. Examination showed a small cauliflower growth at the lower end of the rectum. A colostomy was performed but death ensued on June 11, 1916. A microscopic examination was not obtained.

A daughter, Sophie R., age 24 began in 1913 to lose weight and complain of frequent bowel movements accompanied by a griping pain in the region of the transverse colon. She also noted blood in the stool. In June, 1914, an appendicostomy was performed with plication of an incompetent ileocecal valve. She gained weight and the diarrhea was controlled, but traces of blood were still present. A tuberculin test was positive. In September, 1914, a rectovaginal fistula developed and a large nodular mass was felt in the lower rectum. A microscopic examination showed a malignant adenoma. Radium was used but death ensued on March 30, 1916, at the age of 30 years.

A daughter, Bertha R., age 25, noted a mass the size of a grapefruit in her abdomen. On June 3, 1915, the terminal ileum, cecum and a retroperitoneal fibroma were removed. In June, 1931, she reported that for several months she had noticed a slight change in her bowel action. First there was a diarrhea and now for two weeks there have been gas and distension. Her age is now 40. Examination showed a hard distended abdomen and a circular cauliflower mass, admitting only the tip of the index finger, was felt three inches up the rectum. A colostomy was performed. Microscopic examination of the growth showed an adenocarcinoma. Death occurred on the day following operation.

A son, William R., age 31, in October, 1930, stated that six months previously he had suffered from several attacks of indigestion accompanied by chest pain and gas in the stomach. One month later looseness of the bowels ensued and pain in the left loin with much intestinal gas. Examination showed a large nodular growth of the rectum forming a complete circle. There were also metastases in the lung. On August, 9, 1931, a colostomy was performed. Microscopic examination showed an adenocarcinoma. He died April 1, 1932.

A daughter, Emma R., age 47, the sole surviving member of the family, was admitted to the Presbyterian Hospital, New York, on December 10, 1935. She had had a mucous colitis for many years. Three months before admission her bowel movements, which had previously been two or three a day, changed to six or eight a day. Only once had she noticed a little blood in the stool. Examination showed a mass 10×5 cm. about the umbilicus. There was also a large fungating mass 4 cm. inside the anal sphincter, involving the anterior and left side of the rectal wall, with partial obstruction. A colostomy was performed. Microscopic examination showed carcinoma of the rectum, with metastases to the liver and mesentery. She died on December 28, 1935.

Environment has been considered a factor in the production of cancer. Various members of this family lived at seven different known addresses.

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BRIEF COMMUNICATION AND CASE REPORT

TOTAL GASTRECTOMY *

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Case Report.—H. D., white, male; 35 years old; pharmacist. Admitted to St. Peter's Hospital, Brooklyn, New York, August 7, 1935. He complained of pain in the epigastrium and upper left abdominal quadrant, which occurred in paroxysms radiating upward and to the left. He had suffered this pain for about a year with intervals of abatement rather than remission. He had a sense of fulness whenever he ate solid or semisolid food. Vomited two to three times a day. Had noticed that his stools were streaked with blood and had lost 35 pounds in the last three months. He gave a history of treated lues four years previously but no other serious illness.

Examination of his abdomen revealed a palpable mass in the left upper quadrant, smooth and movable.

Roentgenologic Examination.—Inconclusive. An extensive polypoid new growth involving the upper and middle portion of the stomach was visualized but was not interpreted as malignant; rather, a luetic origin of the lesion was suggested. He was put on active antiluetic therapy and sent home. October 24, 1935, he was readmitted in somewhat worse condition than when he was discharged on August 24, 1935. Pain and vomiting had become aggravated and more tarry stools were evident. Roentgenologic studies showed some advance in the infiltrative lesion of the stomach and after a period of preparatory gastric lavage he was operated upon December 11, 1935.

Under open ether anesthesia an incision was made from the ensiform to the navel along the median side of the left rectus. The cartilages of the tenth and ninth ribs were cut without wounding the pleura and the stomach exposed. An infiltrating growth had invaded the fundus, the posterior wall and the cardiac portion of the lesser curvature. It was thought, however, that a narrow zone distal to the cardia was uninvolved and that an anastomosis between this section of the cardiac end of the stomach and the jejunum could be accomplished. A few enlarged lymph nodes were felt in the gastrocolic omentum. The liver was apparently free of malignant invasion. No adhesions were found other than a doubtful thickening between the fundus of the stomach and the spleen. The pylorus was clamped, divided, and the distal end sutured and inverted with a double purse string suture of chromic gut. The vessels of the greater curvature were clamped and the stomach freed from the colon. In freeing the adhesion to the hilus of the spleen, branches of the vein were wounded and it was found desirable to remove the spleen. In exploring the lesser curvature it was found that the infiltration of the neoplasm approached so close to the cardia that no gastric anastomosis could be accomplished. The esophagus therefore was clamped and divided close to the gastric wall and the stomach removed. A stout silk ligature was applied about the esophagus

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in the bite of the clamp. The transverse mesocolon was opened and a loop of jejunum immediately distal to its beginning was drawn up and secured to the diaphragm posterior to the esophageal hiatus, for a distance of three inches, by a running suture of silk. Stay sutures of silk were introduced lateral to this continuous suture. The jejunum was then opened with a cautery and the ligated end of the esophagus placed into it and secured by three interrupted silk sutures well proximal to the site of its occlusive ligature. The line of continuous sutures which effected the apposition of the jejunum and the diaphragm was now continued around and in front of the esophagus, and a few supporting interrupted sutures were placed in front of this. The ligated esophagus was thus introduced into a loop of jejunum which had been firmly secured to the diaphragm around the site at which the penetration had been effected. The esophagus lay without tension, ligated within the jejunal lumen. The mesenteric opening was secured to the displaced loop of jejunum. No jejunojejunal anastomosis was made. A jejunostomy, employing a No. 16F rubber catheter, was made after Witzel's method, a fold of omentum sealing the approximation to the abdominal wall, the catheter was drawn through a stab wound at the outer edge of the left rectus, two inches above the line of the umbilicus, and the median wound closed in layers without drainage.

A transfusion of 500 cc. of blood was given four hours after the operation. The patient was kept under morphine for three days. He was fed immediately through the jejunostomy tube with peptonized milk and salt solution. He received 2,000 cc. of milk and 1,000 cc. of normal salt solution on his third day and so fed in this summer until the seventeenth day. He was allowed to chew gum. On the fifteenth postoperative day he noted a regurgitation of fluid into his throat but was able to swallow it again. It seemed probable that the ligated esophagus had opened, as it was hoped would eventuate, and on the seventeenth day feeding by mouth was begun. This has been continued with increasing courage since that time. He is now eating cereals, soups, eggs and soft vegetables with chopped meat.

The pathologic examination showed an ulcerating infiltrative lesion of the stomach—an adenocarcinoma—involving all the muscular layers with many areas of myxomatous degeneration. Lymph nodes showed malignant invasion.

This case is reported because of the following points of interest:

(1) A method of esophagojejunal anastomosis is submitted which depends upon suture of the intestine to the diaphragm, the ligated esophagus being introduced into the intestinal lumen within that suture ring.

(2) A secondary jejunostomy, for nutrition, maintained the patient's weight and well being for 17 days, until the ligature of the esophagus had cut through; after continuity had been established, feeding by mouth was begun and maintained.

(3) The presence of ravenous hunger in the complete absence of the stomach is noted—without comment on present physiologic dicta.

(4) The absence of blood changes, up to this time, is noted.

(5) The fact that the spleen was removed is also noted, and the possibility that damage to the circulation of that organ may have played a part in some fatalities following total gastrectomy should not be ignored.

DISCUSSION.—DR. RICHARD LEWISOHN (New York) congratulated Doc-

tor Jennings especially on the fact that, to his knowledge, this was the first case surviving a total gastrectomy to be presented before the New York Surgical Society. Doctor Lewisohn wondered in how many instances Doctor Jennings's original procedure would be applicable, and said that one might well speculate on what would happen should the ligature fail to cut through properly. The procedure would seem to be applicable only in cases with a fairly long intra-abdominal esophagus. Doctor Lewisohn described a case in whom he had performed total gastrectomy in December, 1935, and stated that the patient died, due to a leak, within four weeks. The prognosis in total gastrectomy, especially for cancer, is not good. If the patient survives the operation, he succumbs, usually within a short period, of recurrence. He wished to inquire whether the microscopic sections of the cardiac end of the stomach showed esophageal mucosa.

Dr. Carl Eggers (New York) said that as he understood it, Doctor Jennings's operation of total gastrectomy had not been deliberately planned, but was in the nature of an experimental operation. That he had met the conditions found at operation in the brilliant way in which this was done was one of the most noteworthy features of his presentation. Total gastrectomy is a formidable operation, it is rarely attempted, and, when successfully performed, frequently fails as the result of some complication. Leakage with a fatal peritonitis is the usual cause of death.

There are two methods of anastomosis most frequently employed; an esophagoduodenostomy, or an esophagojejunostomy. The former is always subjected to great tension or there may be interference with the blood supply, both of which favor an insecure anastomosis. Doctor Jennings employed the latter method and emphasized two principles in its performance. He fastened the jejunum securely to the under surface of the diaphragm, thereby avoiding tension, and he kept the esophagus stump tied with a silk ligature until it cut through spontaneously, thereby preventing the entrance of organisms from the mouth and keeping the anastomosis at rest until it was healed. A jejunostomy below the anastomosis was used for feeding.

There are several points of interest connected with this case, the most important of which seems to be the feasibility of implanting an esophagus stump, devoid of a serous covering, into an organ which has a peritoneal coat. This problem has interested all surgeons who perform resections of the cardia or of the lower esophagus for carcinoma. Doctor Fischer of this society has described a method of implanting the esophagus stump into the fundus of the stomach after resection of the cardia, which involves the same principles emphasized by Doctor Jennings. He has also advocated leaving the stump closed and permitting the ligature to cut through at a later date. The principle seems sound, as it will effectually put the anastomosis at rest, and at the same time prevents mouth organisms from coming down. That healing of an uncovered esophagus stump into an organ covered with peritoneum can take place is proved by the case presented tonight. Personally, I have a beautiful speciman illustrating this point. After resecting a carcinoma of the cardia from above, I had implanted the *open* esophagus stump into the fundus of the stomach. The patient died ten days later as the result of a small mediastinal abscess. The suture line was intact, the coats firmly united. Soiling of the field had apparently occurred at the time of operation and might have

been avoided by implanting the *closed* stump into the stomach. In case one fears that a silk ligature may not cut through one may use chromic catgut, which would dissolve in a reasonable time.

CORRECTION

In the title of the article "Sacrococcygeal Transrectal Approach for High Rectovaginal and Vesicovaginal Fistulae" by Dr. John C. A. Gerster which appeared in the ANNALS OF SURGERY, Vol. 104, No. 2, pp. 244-247, August, 1936, the word Vesicovaginal should be corrected to Rectovesical.

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